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TIDAL DRAINAGE OF THE URINARY BLADDER*

A Preliminary Report of This Method of Treatment As Applied To "Cord Bladders" With a Description of the Apparatus

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ABNORMAL motility of the urinary bladder presents certain special problems in therapeusis, the solution of which has been notoriously unsatisfactory. This has been particularly true in those cases in which the innervation of the viscus has been altered. This causes on the one hand paralysis or weakness and on the other overactivity and hypertrophy of the detrusor muscle, and the internal and external sphincters. Ideal treatment under such circumstances calls for a minimum of catheterization, provision for control of intravesical pressure and therefore the degree of distention of the bladder, and a method whereby irrigation together with the complete removal of residual urine can be carried out. This paper comprises a descripton of an apparatus that in our hands has fulfilled these requirements. A brief preliminary report of its use as a therapeutic agent in the treatment of certain cases of so-called cord or neurogenic bladders is also included.

Tidal drainage has been previously described in relation to both the treatment of pleural empyema and that of urinary bladder disease. Its characteristic is or should be an automatic alternate ebb and flow of the fluid content of a closed cavity. For this reason we do not class the treatment described by Hart[1,2] as true tidal drainage. Automaticity is replaced in his original apparatus by a pumping action obtained by the motion of the chest wall and in the later models by a suction pump. The only apparatus which has provided tidal drainage in bladder disease is that developed by Laver some time previous to 1929 and refined in 1934[8]. We have been unable to find his original description except as given in the Down Brothers Instrument Catalogue of 1929[7]. By using one of his earlier apparatuses as a model and also in actual practice we developed a similar but less expensive machine which was described by one of us also in 1934[14]. We believed that all three of these were efficient until more accurate observations made necessary in an attempt to attach a manometer demonstrated that a part only of the bladder contents was removed at each "ebb". Although this imperfect apparatus was a great improvement on any of the commoner methods now in use, the problem of residual urine was still unsolved. Other types of apparatus such as described by Young[6] and Davis[5], do not produce tidal drainage as described above. Their action is limited to the decompression of a distended bladder either with or without additional irrigation.

Our present apparatus, to be described in detail below, alternately fills the bladder to a predetermined height of intravesical pressure and then empties it by a combination of siphonage and gravity flow, the siphon being interrupted coincident with complete evacuation. This process is dependent upon three fundamental factors. These are first, the introduction into the system of a small reservoir equipped with an air vent which receives the irrigating fluid as it drips from the container. Secondly, an arrangement whereby the siphon comes off between the reservoir and the bladder, and thirdly, providing the tubing through which the bladder empties with a diameter at all points at least twice that of the tubing through which the reservoir empties. (Figure 1.)

The different parts are as follows: A 1000 cc. container graduated in 25 cc. amounts which empties through the bottom is attached by a 30 cm. piece of rubber tubing to a dropper which must be without an air vent. The rate of flow through the dropper is controlled by an adjustable pressure clamp placed above it. The lower end of the dropper is attached by an appropriate length of rubber tubing to the upper end of the vertical limb of a glass T tube set on end and having an internal diameter of 4 mm., the other end of which leads to the reservoir. The reservoir is a ½ pint preserve jar with a lock-bar top. This can be bought at any Woolworth 5 and 10 cent store. A No. 9 rubber cork with two perforations closes and is held solidly

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in the bottle with the lock-bar. Through the perforations are inserted two pieces of glass tubing with an inside diameter of 3 mm. Of these one reaches to the bottom of the jar and is attached to the lower end of the vertical limb of the T tube in the container system. In addition the tip of this piece of tubing which is inside of the reservoir is fused to a diameter of 2 mm. or less. Through the other perforation a similar piece of tubing is inserted. This reaches only to the bottom of the cork and

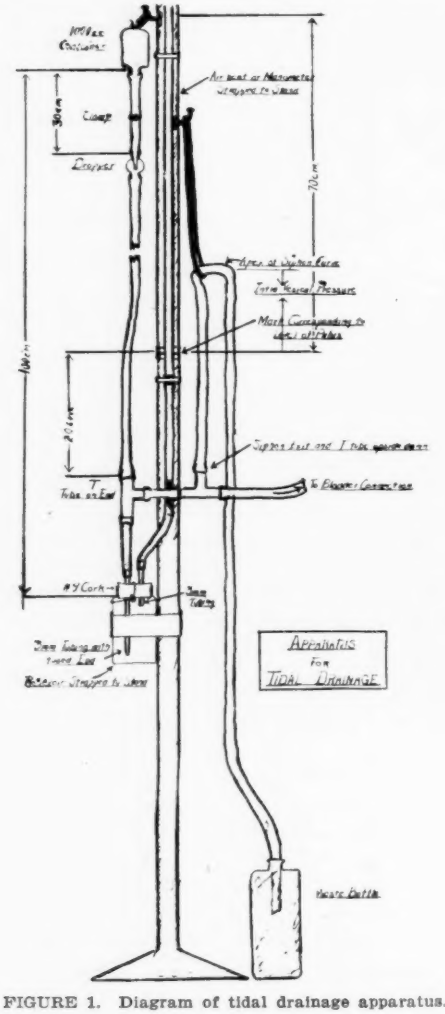

FIGURE 1. Diagram of tidal drainage apparatus.

neither end is decreased in size. The upper end is attached through a stiff-walled rubber tube 18 cm. long to a straight glass tube 70 cm. long. If the apparatus is to be used only for tidal drainage, this latter should have a diameter of 7 mm. If however it is desired to measure bladder pressures, the bore should be 3 mm. and a centimeter scale should be placed in back of it when the apparatus is set up. In either event the primary purpose is to provide an air-vent for the reservoir. The horizontal limb of the T tube on the container set-up is attached to one end of the horizontal limb of a similar T tube placed upside down. From the other end a connection is made to the catheter in the bladder. A rubber tube leads from the vertical arm of this last T tube upwards for a predetermined distance and then downward to a waste bottle.

Photograph of tidal drainage apparatus in use.

The apparatus is set up as follows: (Photo No. 1.) A hat stand or irrigator stand is marked in some suitable way at a point level with the pubis when the patient is flat on his back in bed. This is a sufficiently accurate and accepted 0 point from which to measure vesical pressure. The reservoir with the container and siphon sections attached to it is then fastened by adhesive plaster to the stand at a point which will bring the joined horizontal limbs of the two T tubes 20 cm. below this mark. This permits of a margin of safety for excessive respiratory exeursions of the fluid in the system. These might otherwise activate the siphon before the desired intravesical pressure had actually been attained. The container with its attachments is hung from the stand at least 100 cm. above the reservoir. The air vent or manometer, as the case may be, is now attached by its rubber tubing to the reservoir and then the glass strapped to the stand with adhesive plaster. The lower end of

this glass tubing should be at or just below the mark indicating the level of the pubis. The siphon rubber tube is now led upward to a predetermined number of centimeters above the level of the pubis and then curved downward without kinking to end in the waste bottle. The height in centimeters of this curve above the level of the pubis will be the level of the intravesical pressure at which the bladder will be emptied. The tube may be curved without kinking by splitting lengthwise a two-inch piece of the same type tubing and using it to re-enforce the curve on its lower side, the whole being supported by a bandage tied to an appropriate hook on the stand. The catheter connection is made and the flow started through the dropper. Providing all joints are air-tight the apparatus will now work.

In a typical cycle the reservoir is first filled to the level of the horizontal arms of the T tubes after which the fluid flows into the bladder. During the filling the air escapes through the air vent. As the bladder fills, the intravesical pressure rises even if there is complete atonia. Coincident with that rise the fluid in the container system below the dropper, in the air vent and in the part of the siphon leading upwards, rises simultaneously and equally. This rise continues until the top of the curve of the siphon tube is reached (and corresponding levels in the air-vent and the container system). At this point the intravesical pressure is equal to the distance in centimeters between the level of the pubis and the apex of the siphon curve. The addition of more fluid overflows the siphon which because of its position empties the reservoir and bladder simultaneously. The bladder empties via the catheter and bladder end of the siphon T tube; the reservoir via the glass tubing through which it had been filled, the T tube of the container system and the container end of the siphon T tube. However, because the size of its discharge tube is ½ the size or less of that of the bladder, the reservoir empties only ½ as rapidly. Fluid remains in the air-vent until both are empty thus sealing it and permitting the siphon to continue. With the emptying of the reservoir, usually well after the bladder, the air-vent empties also and air then enters through this latter, rushes up into the siphon and interrupts the flow. The stage is now set for a repetition. Should the bladder be so large as not to empty as described, the residual urine will not remain in the bladder. It will drain by gravity into the reservoir, the cycle will be hastened and such excess urine will be removed. The apparatus is working perfectly when respiratory excursions are visible in the air-vent, there is no leak around the catheter, the air enters only by the route described above and the jar empties completely at each cycle. The catheter should be removed, cleaned and replaced once a week. This apparatus is cheap and simple to construct. When set up it acts automatically for an indefinite period with a minimum of attention. Furthermore, observations at present being carried out, but not as yet completed, indicate that manometric studies of intravesical pressures and, by inference, detrusor muscle activity can be made with sufficient accuracy with this apparatus to indicate in some detail what progress the bladder is making toward eventual recovery. These measurements also provide a more accurate diagnostic estimation of the cause of the abnormal bladder activity and furnish a true estimate of the amount of residual urine.

Bladders suitable for treatment by tidal drainage are preëminently those associated with spinal cord injuries. Other methods of treatment such as those commonly in use to-day are recognized as unsatisfactory. For example repeated urethral drainage or an inlying catheter lead to urinary tract infection with possibly considerable damage to the bladder wall from overdistention. Attempts to produce so-called automaticity by allowing the bladder to empty itself either with or without the aid of external pressure certainly damage the bladder wall and probably lead to infection. In either case such infection retards the return of segmental reflex activity and usually leads to death. Suprapubic drainage, like watchful waiting, condemns the patient to a greater liability to decubitus ulcerations on account of the leakage and resultant constant wetting of the bed. Shrinkage of the bladder is also prone to follow. Even tidal drainage carries possibilities of harm in its wake if it is misused. Such therapy results directly from ignorance of the physiology of micturition and the changes that take place following a spinal cord injury.

For a complete review of the knowledge available to date in regard to these problems, the works of Holmes[13], Learmonth[10], Fulton[9], Denney Brown and Graeme Robertson[11, 12] should be consulted. (Figure 2.) Suffice it to say here that normal micturition is a reflex act stimulated by distention of the bladder wall associated with alternating periods of stretch and relaxation. This results in contraction of the detrusor muscle with reciprocal relaxation of the internal and external sphincters. These activities are mediated by the parasympathetic and pudic nerves. Emptying may be inhibited by impulses from higher centres which short circuit the motor side of the reflex arc or by impulses from the same source which prevent the relaxation of the external sphincter. Additional inhibitory impulses through the sympathetic nerve supply are possible but probably of no practical significance. The sensation of pain on the other hand undoubtedly is carried over this latter connection. Facilitation of emptying is produced by failure to inhibit.

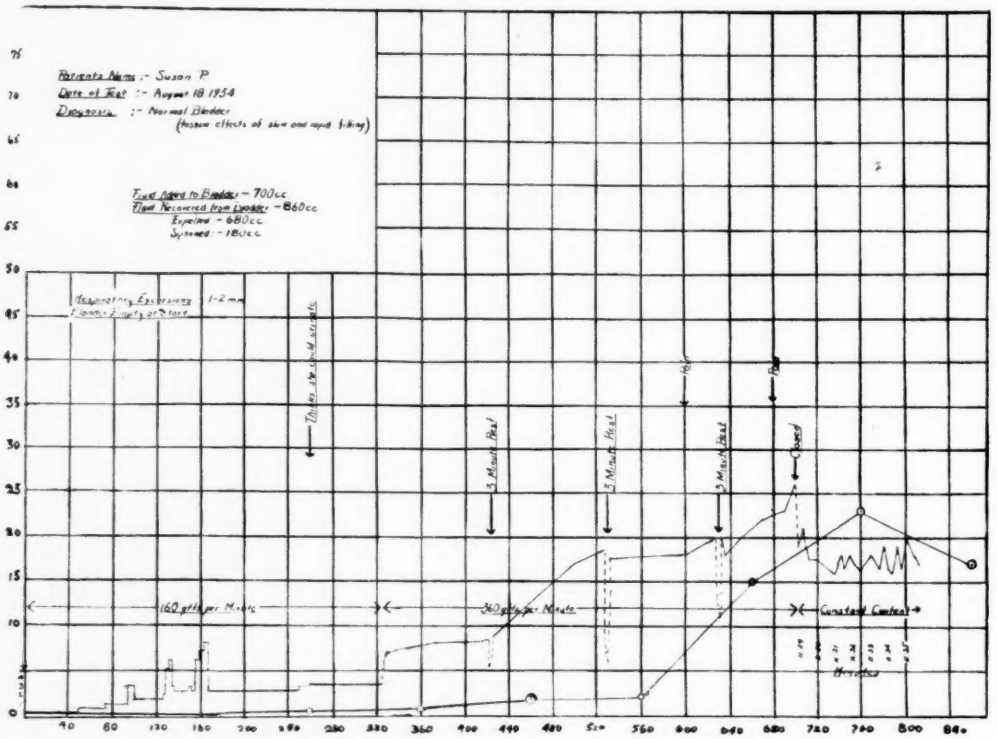

FIGURE 2. Graph of activity of normal bladder as affected by a variation in the rate of filling. Abscissa shows cc. of fill and ordinate cm. of pressure.

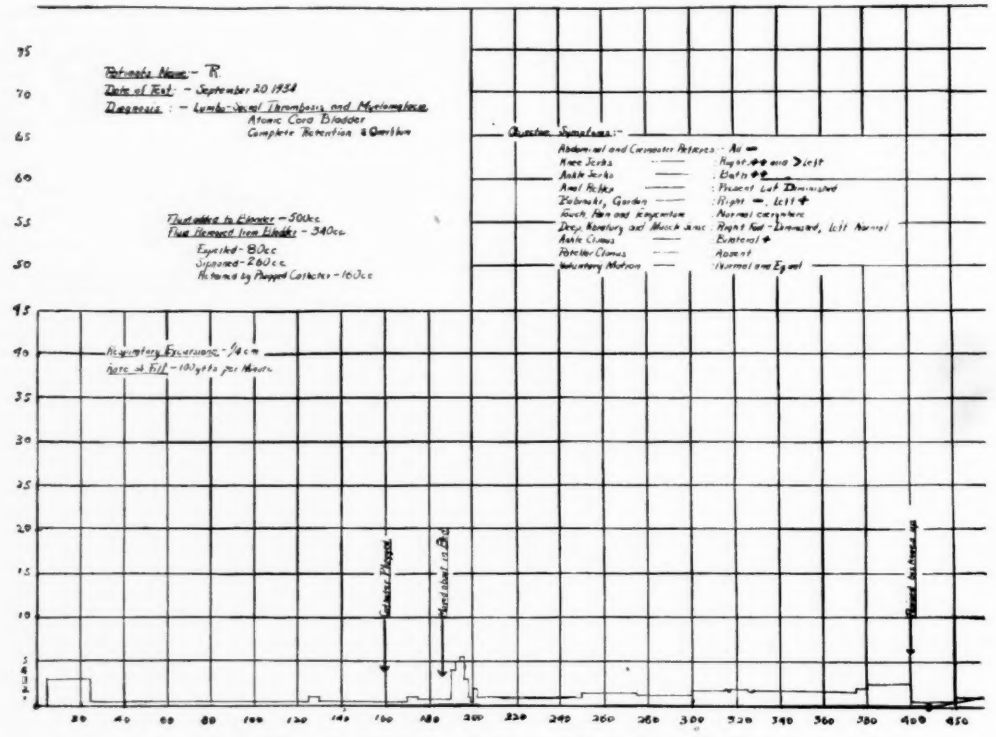
Patient's Name - R.
Date of Test - September 20 1934
Diagnosis : - Lumbo-Sacral Thrombosis and Myelomalacia
Atonic Cord Bladder
Complete Retention & Overflow
Fluid Injected to Bladder - 500cc
Fluid Recovered from Bladder - 340cc
Expelled - 80cc
Syphoned - 260cc
Returned by Pumped Catheter - 160cc
Respiratory Excursions - 1/4 cm
Rate of Fill - 100 gtts per Minute
Catheter Plugged
Fluid at Level of Bed
Patient Catheterized
Centimeters
Minutes

FIGURE 3. Graph of activity of "Atonic Cord Bladder". Complete retention with overflow from overdistention. Abscissa shows cc. of fill and ordinate cm. of pressure.

Alterations in bladder physiology following spinal cord damage not only remove the organ from the inhibitory influence exerted by higher centres but materially alter the reflex itself. Unless this is understood, treatment cannot be intelligently applied no matter what type is used. Cord bladders are neither "paralyzed" nor "automatic". Their degree of activity is a direct function of the degree of interference with the reflex response to a given intravesical stimulus. This is measurable in terms of contractility of the bladder wall and external sphincter and divides these cases into four groups.

The "atonic cord bladder" forms the first of these groups. (Figure 3.) Following all cord injuries whether transection, contusion, concussion, hematomyelia or edema, there is a period of spinal shock. While this phenomenon lasts, the bladder reflex like all segmental spinal reflex activity becomes inhibited or temporarily destroyed. A characteristic of this inhibition is complete atonia of the detrusor muscle. A reciprocal contraction of the internal sphincter is associated with this lack of tone. Retention of urine and overdistention of the bladder result. If unrelieved, this in its turn produces a varying degree of permanent damage to its muscle fibres, residual urine and spreading infection. If the infection assumes major proportions before the period of spinal shock is past, this in itself continues the areflexia until death or recovery at an unduly late date ensues. Tidal drainage not only prevents this distention but provides regular irrigation and alternate stretch and relaxation of the bladder wall under a controlled amount of intravesical pressure.

Following recovery from the spinal shock and provided no major infection has set in, the "atonic cord bladder" becomes an "autonomic cord bladder". This is particularly evident in patients in whom the spinal cord has been transected, though this type of bladder is probably not necessarily limited to this type of cord injury. This forms the second group of cord bladders. (Figure 4.) Reflex activity returns in the form of inefficient detrusor contractions which are probably mediated by the autonomic neural plexus in the bladder wall itself[12]. These contractions open the internal sphincter (the external being still flaccid), but fail to expel any urine. A leaky overflow bladder results. Tidal drainage keeps such a patient dry and permits the weak bladder contractions to exert their maximum effect and thus aid in the return of the more efficient spinal reflex mechanism. (Figure 5.)

Further bladder activity resulting from a more complete recovery of the cord injury depends on the type of original injury and the level of maximum damage. The so-called "cord bladder" is commonly associated with transection, and discussion at this time is limited to such cases. They make up the remainder of

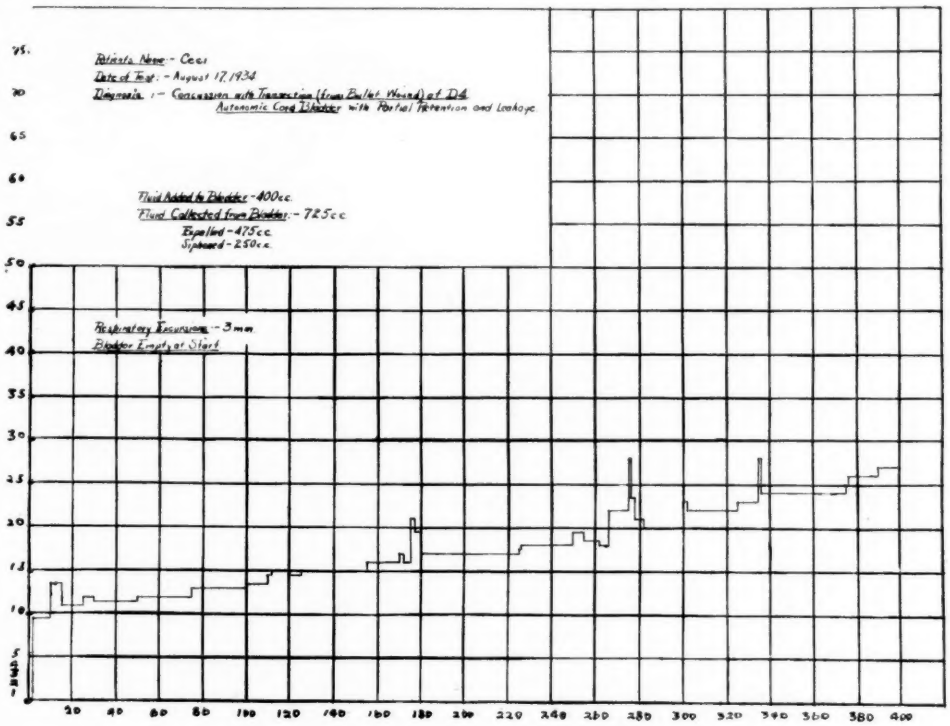

FIGURE 4. Graph of activity of "Autonomic Cord Bladder" 10 days after injury and before institution of tidal drainage. Retention with leakage and residual urine. Abscissa shows cc. of fill and ordinate cm. of pressure.

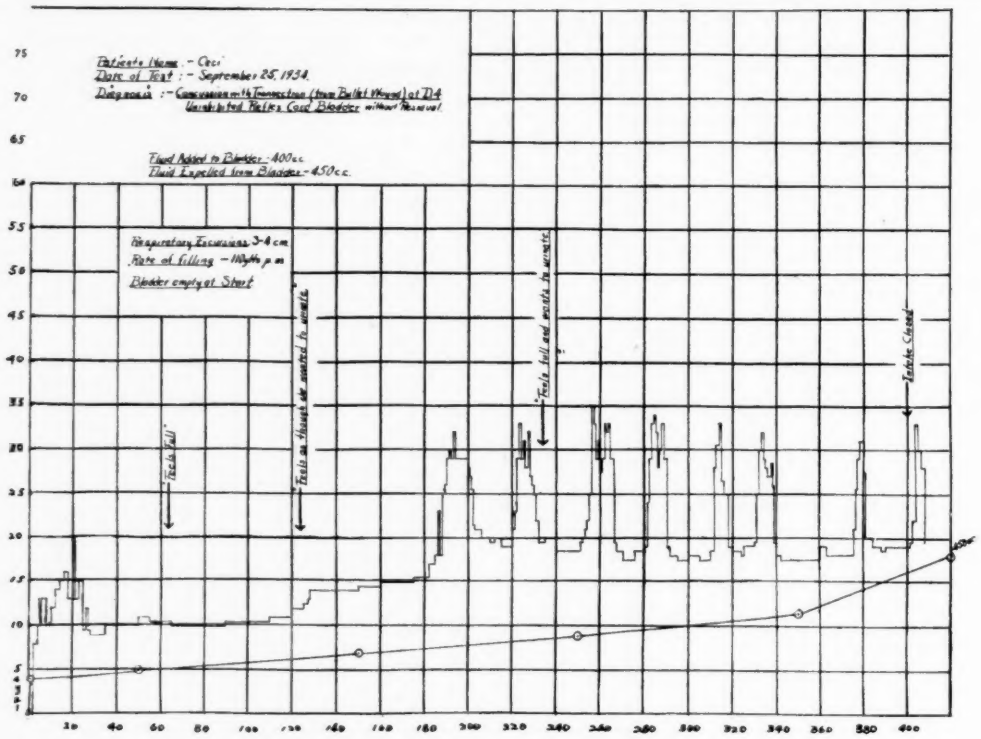
Patient's Name :- Cecil
Date of Test :- September 25, 1934.
Diagnosis :- Concussion with Transection (from Bullet Wound) at D4
Uninhibited Reflex Cord Bladder without Retention
Fluid Added to Bladder - 400cc.
Fluid Expelled from Bladder - 450cc.
Respiratory Excursions 3-4 cm.
Rate of Filling - 100cc p.m.
Bladder empty at Start
Feels Full
Feels as though she wanted to urinate
Feels full and wants to urinate
Feels Closed

FIGURE 5. Graph of activity of same bladder as in Figure 4, after 5½ weeks of tidal drainage. Bladder now "uninhibited reflex" type. No retention, no leakage and no residual. Complete emptying by means of unaided bladder contractions. Abscissa shows cc. of fill and ordinate cm. of pressure.

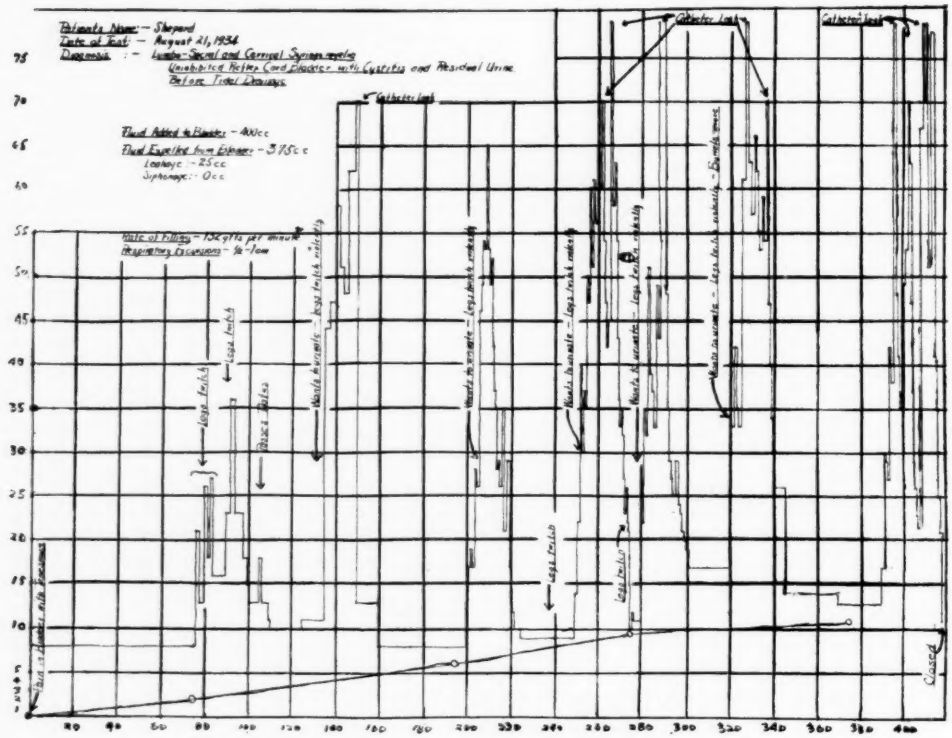
Patient's Name :- Shepard
Date of Test :- August 21, 1934
Diagnosis :- Lumbo-Sacral and Cervical Spinal Injuries
Uninhibited Reflex Cord Bladder with Cystitis and Residual Urine
Before Tidal Drainage
Catheterized
Fluid Added to Bladder - 400cc
Fluid Expelled from Bladder - 375cc
Leakage - 25cc
Siphonage - 0cc
Rate of Filling - 150 cycles per minute
Respiratory Excursions - ½-1cm
Legs Twitch
Bladder Reflex
Wants to urinate - Legs twitch violently
Catheter Left
Catheter Pulled
Bladder empty - Legs twitch violently
Wants to urinate
Closed

FIGURE 6. Graph of activity of inefficient "Uninhibited Reflex Cord Bladder" with cystitis, toxemia, tenesmus and residual urine. Abscissa shows cc. of fill and ordinate cm. of pressure.

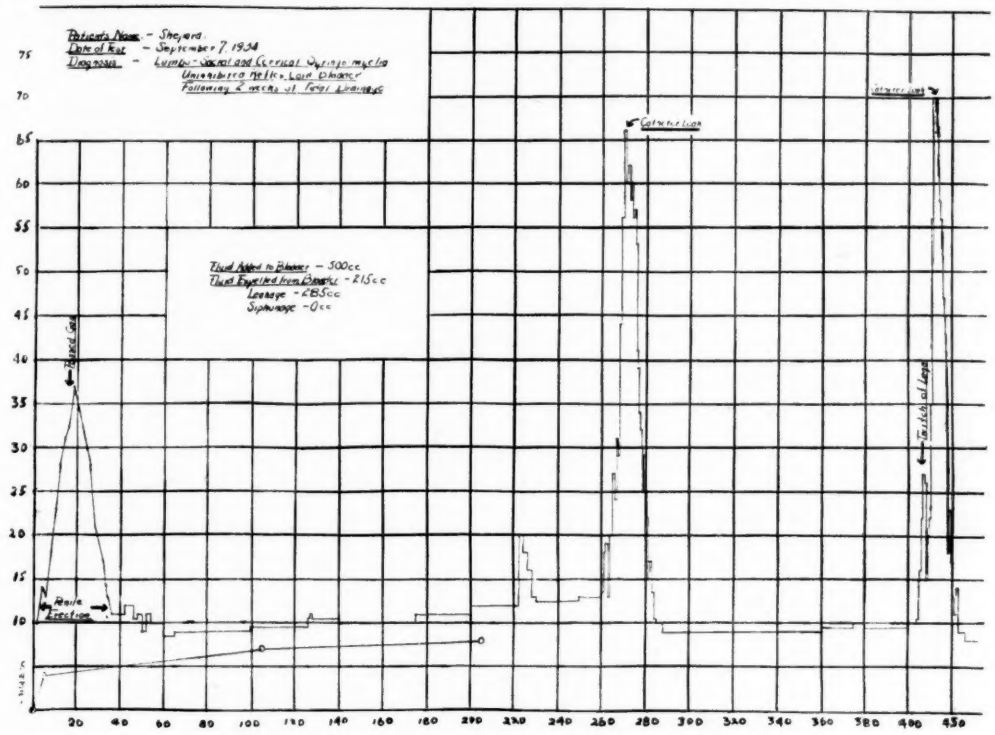
Patient's Name - Shepard
Date of Test - September 7, 1934
Diagnosis - Lumbo-Sacral and Cervical Syringomyelia
Uninhibited Reflex Cord Bladder
Following 2 weeks of Total Drainage
Catheterized
Fluid Added to Bladder - 300cc
Fluid Expelled from Bladder - 215cc
Leakage - 285cc
Siphonage - 0cc
Tonic Cord
Reflex Erection
Twitch of Legs

FIGURE 7. Graph of activity of the same bladder as in Figure 6, after 17 days of tidal drainage. "Microscopic Cystitis" only, normal temperature for 10 days, no tenesmus and no residual urine. Abscissa shows cc. of fill and ordinate cm. of pressure.

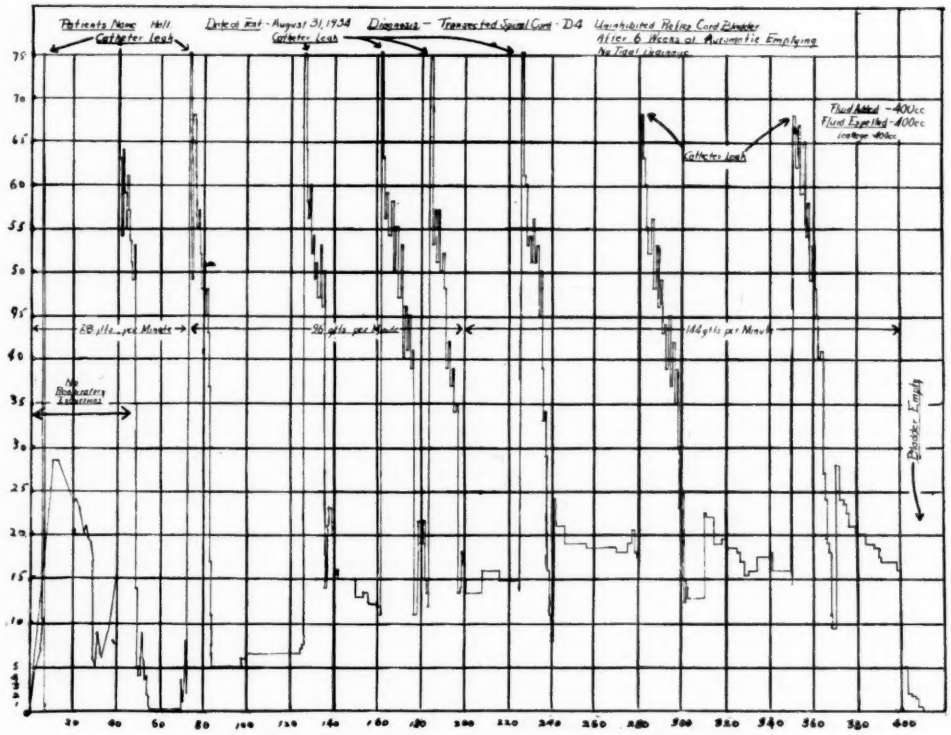

FIGURE 8. Graph of so-called "Automatic Cord Bladder" which is actually an overactive inefficient "Uninhibited Reflex Cord Bladder". Unduly frequent evacuation with constant "wetting" and marked shrinking of bladder capacity. No cystitis. Abscissa shows cc. of fill and ordinate cm. of pressure.

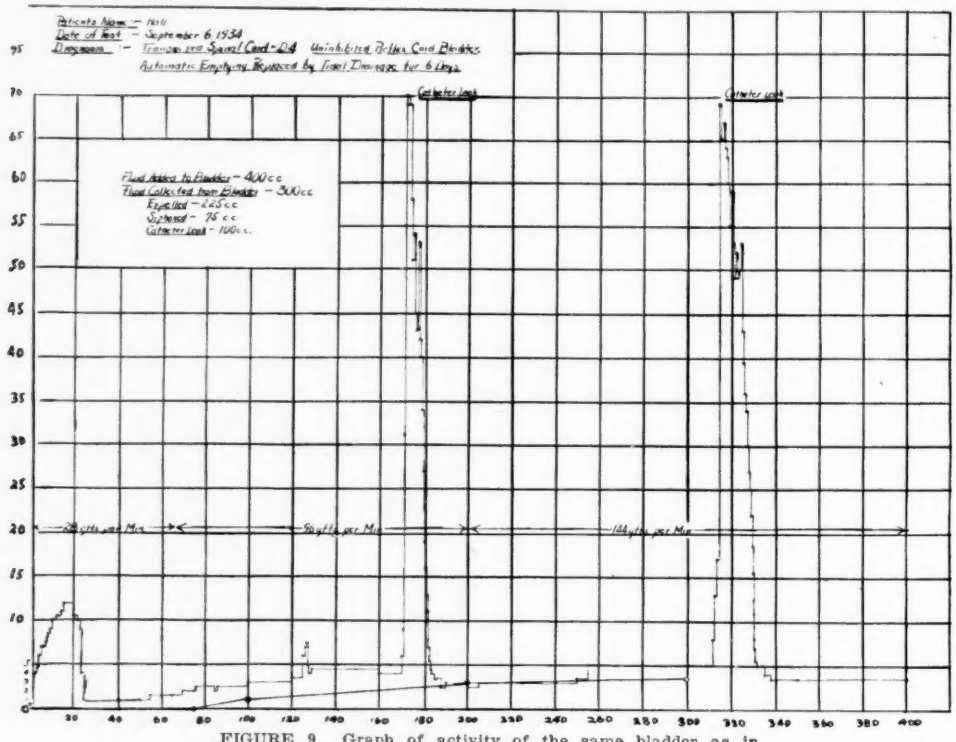
Patients Name – Nov
Date of Test – September 6 1934
Diagnosis – Transected Spinal Cord – D4 Uninhibited Reflex Cord Bladder
Automatic Emptying Replaced by Tidal Drainage for 6 Days
Catheter Leak
Catheter Leak
Fluid Added to Bladder – 400 cc
Fluid Collected from Bladder – 500 cc
Expelled – 425 cc
Siphoned – 75 cc
Catheter Leak – 100 cc.
28 gtts per Min
84 gtts per Min
144 gtts per Min

FIGURE 9. Graph of activity of the same bladder as in Figure 8, following 7 days of tidal drainage. Normal rate of contractions and bladder capacity. Abscissa shows cc. of fill and ordinate cm. of pressure.

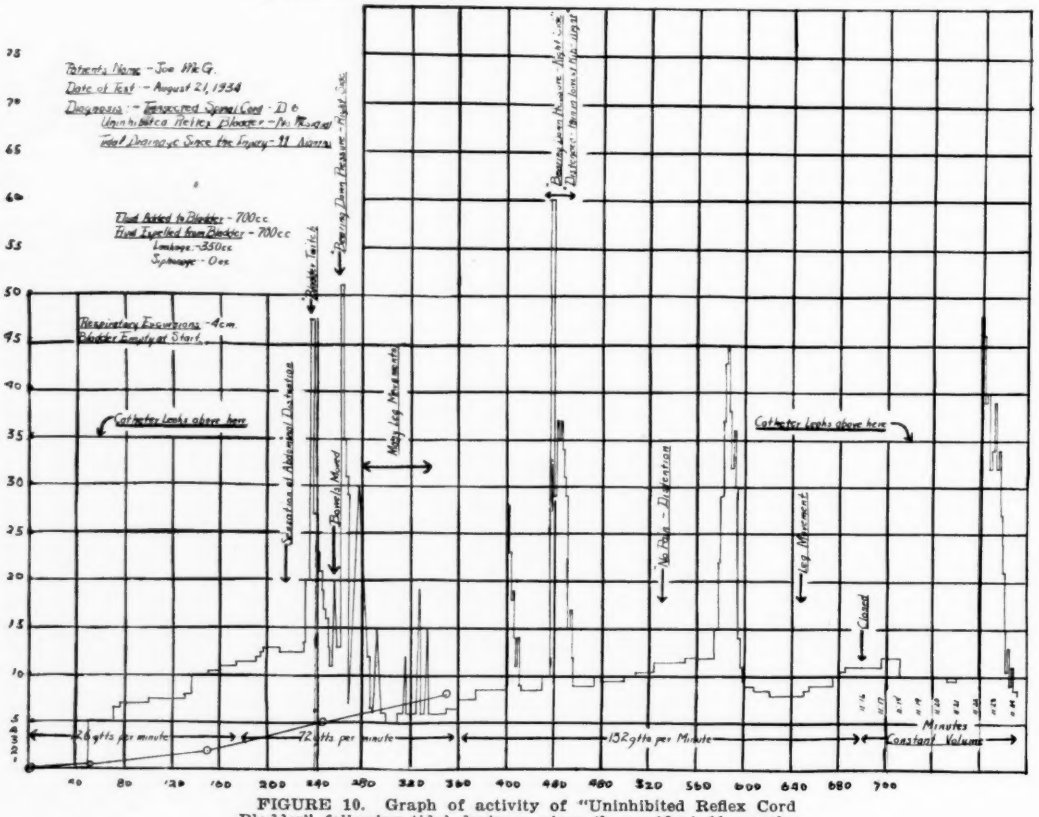
Patients Name – Joe McG.
Date of Test – August 21, 1934
Diagnosis – Transected Spinal Cord – D 6
Uninhibited Reflex Bladder – No Residual
Tidal Drainage Since the Injury – 11 Months
Fluid Added to Bladder – 700 cc
Fluid Expelled from Bladder – 700 cc
Leakage – 350 cc
Siphonage – 0 cc
Respiratory Excursions – 4 cm.
Bladder Empty at Start
Catheter Leaks above here
Sensations of Abdominal Distention
Painful Muscle
Many Leg Movements
No Pain – Distention
Leg Movement
Closed
Catheter Leaks above here
Minutes
26 gtts per minute
72 gtts per minute
132 gtts per Minute
Constant Volume

FIGURE 10. Graph of activity of "Uninhibited Reflex Cord Bladder" following tidal drainage since the accident 11 months previously. Repeated cystoscopies, ureteral catheterizations and pyelograms show that the genito-urinary tract is completely normal except for a mild cystitis. Bladder empties completely unaided. There is no residual urine and the capacity of the bladder is normal. Abscissa shows cc. of fill and ordinate cm. of pressure.

our four groups and commonly go under the misnomer of "automatic bladders". Their characteristics depend upon the level of transection.

If the injury be one that destroys the sacral spinal segments or otherwise separates anatomically the bladder from its neural connections with the central nervous system, an "hypertonic cord bladder" develops. This is a further development of the "autonomic cord bladder" described above. In it a more efficient motor response is produced as a result of a stretch stimulus of the bladder wall and the internal sphincter not only opens but the detrusor contracts sufficiently to expel the contents. We have had no personal experience with this particular type of bladder but it is said[12] that, as the result of unbridled reflex autonomy, the muscular wall hypertrophies and the bladder becomes incapable of holding more than a small amount of urine. In these patients the external sphincter is flaccid and there is no anal reflex. We suggest that a measure of control of the incessant stimulus and contraction as well as prevention of shrinkage may be accomplished through the use of tidal drainage set to empty the bladder at a high intravesical pressure.

If on the other hand the transection is above the sacral segments, another type of automatic bladder is established. In its final form, this is a normal bladder which has been deprived of the inhibitory effect of higher centres. During the first stages of bladder recovery following such an injury, the activity of the detrusor muscle is set off by a stretch stimulus that is close to normal for that particular viscus. The variation from the perfected end-result in this inefficient automatic cord bladder lies in the incomplete contractions of the detrusor. Partial emptying and consequent residual urine follow. Such a situation may go unrecognized in the face of an apparent "automaticity" and cystitis (Figures 6 and 7) and infection of the urinary tract develop. This may cause loss of all reflex activity and a reversion to the atonic state as seen with spinal shock. Tidal drainage reduces the likelihood of this regression. Residual urine cannot collect, the discharge siphon pressure can be set at the optimum level and the bladder given its best chance to become a truly efficient normal reflex mechanism again. (Figures 8 and 9.) In these cases the external sphincter is hypertonic but reflexly active and there is an anal reflex present. The activity of the former may be masked, however, by excessive flexor response of a mass reflex nature. This either prevents relaxation of the external sphincter with mechanical retention or may go still farther and cause movement of the anterior abdominal muscles sufficient to compress the bladder. (Figure 10.) This stimulates the detrusor which contracts with a resultant reflex relaxation of both sphincters followed by emptying of the bladder. In either case the presence of a urethral catheter prevents external sphincteric blockade and the additional tidal drainage obviates the infection associated with repeated catheterization or the ordinary type of inlying catheter.

CONCLUSIONS

1. An apparatus for automatic tidal drainage of the paralyzed urinary bladder is described.

2. The physiopathology of the bladder is discussed particularly in association with spinal cord injuries.

3. So-called cord and automatic bladders are divided into four classes and a preliminary report on tidal drainage as a method of treatment for each class is made.

4. The four classes are as follows: "Atonic Cord Bladder," "Autonomic Cord Bladder," "Hypertonic Cord Bladder," and an "Uninhibited Reflex Cord Bladder."

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DISCUSSION

DR. WILLIAM C. QUINBY, Boston, Mass.: It needs a good deal of temerity to rise to discuss such an interesting paper as Dr. Munro has just given us. I think it is important, however, to get clear in our minds one or two points—maybe they are not clear in mine—but in the bladder whose nervous balance has been interfered with, several things may happen, and the bladder will then fall into one of the four categories mentioned by Dr. Munro.

I am not clear that I understand, as regards tidal drainage, whether Dr. Munro is trying to cure a bladder whose nervous mechanism has been upset, or whether by this apparatus he is trying to save life. The two matters are in some respects different. The usual patient with broken back is, I suppose, most apt to die a death of urinary sepsis. This is probably the commonest form of death in accidents involving the spinal cord. I am speaking of trauma rather than of cord tumors. The

question uppermost during the War was, "Shall a patient with a spinal cord injury be immediately catheterized, or not?" There were those who held that immediate catheterization of such a patient merely hastened his death by infection. There were others who took the opposite view, saying that if the bladder were not emptied, it became overdistended, the sphincteric muscles were so spasmodic that the bladder could not be emptied and that its wall becoming inefficient, infection and early death were promoted by such non-interference.

I am not clear in my mind exactly what advantages Dr. Munro ascribes to tidal drainage over constant drainage. The tidal element in his drainage apparatus must be designed to exercise the bladder wall, or at least to give opportunity for the bladder to fill and contract continuously as it does during normal life. This may be a wise procedure, or it may not. I am not sufficiently familiar with this method of treatment to be able to form any judgment in the matter. I do feel, however, that one of the factors in the bladder whose nervous mechanism is upset, does not rest entirely in the bladder itself. I refer to the very important factor of regurgitation of bladder contents upward toward each kidney through the ureters. I have no personal doubt that regurgitation of bladder contents upward takes place under certain circumstances in all of us in complete health. If one studies a series of cystograms made by the roentgenologist to illustrate the outline of the normal bladder, it is easy to note regurgitation of the fluid used to fill the bladder upward into one or both of the ureters. In the past my associates and I have demonstrated experimentally in very definite manner that under certain conditions of balanced intravesical pressure plus the question of irritability of the bladder wall, there may occur, and there can be caused to occur, upward progression of the bladder contents to each kidney. This phenomenon can be seen in bladders in which the ureterovesical valve mechanism is entirely normal. It is this factor in those bladders whose neurogenic balance has been upset, which I feel to be of great importance, even greater than that as regards the absolute integrity of the detrusor muscle.

I should like very much to ask Dr. Munro in closing to tell us whether with this apparatus he is attempting to treat the bladder per se, or, on the other hand, is attempting to prevent death from urosepsis. It is one thing to keep the bladder wall exercised, but it is an entirely different matter to keep it free from infection.

My feeling is that this apparatus will be applied more successfully in those lesions in which the underlying condition in the spinal cord may be susceptible of recovery; as, for instance, in the temporary upset of bladder function during and following enucleation of a cord tumor. That any method of this sort, tidal or not, will prevent urinary infection in the transverse lesions of the cord caused by a broken back, I very much doubt.

DR. DAVID CHEEVER, Boston, Mass.: This isn't any further discussion, but evidently a very careful piece of work on the part of Dr. Munro, has been done. As I am not a graduate in hydraulic engineering, of the Massachusetts Institute of Technology, I don't mind saying that I don't understand fully the apparatus, and I should be very much interested if it were possible and time permitted, to have Dr. Munro's diagram thrown on the screen so that he could take the pointer and show us just how it works.

DR. DONALD MUNRO, Boston, Mass.: (Slide.) The apparatus works because the siphon tube comes off between the bladder and a reservoir which collects the irrigating fluid before it enters the bladder. It also works because the emptying of the reservoir takes place at half the rate of the emptying of the bladder, the size of this tubing here being reduced to half the size of this opening here, or less.

Perhaps it will be more understandable if I go through what happens. The irrigating fluid is placed in the container, and drops through here at any rate one chooses to use, forty to sixty drops a minute, flows down here, fills this reservoir and while it is filling this reservoir, the air escapes through the air vent or manometer. The fluid completely fills the reservoir and then rises in this tubing and this tube to the level here, after which the fluid flows in this direction in through a catheter along this arm of the T-tubes, to enter the bladder.

It will do that whether the bladder is higher or lower than this tubing, provided the pressure is sufficiently high from this point.

After the bladder is full, the fluid from the reservoir, or rather from the container, instead of entering the reservoir or bladder—there is no place else for it to go except up these various tubings,—enters here and here, and in this arm of the siphon, and when it reaches this point, the apex of the siphon, the addition of a small amount of fluid causes the overflow to take place down this tubing, into the waste bottle. That starts a siphon action which draws simultaneously from the reservoir and also from the bladder; however, only half as fast from the reservoir as the bladder, because the outlet is half the size in the reservoir that it is to the bladder. Therefore the bladder empties first, and the siphon is not broken, because until the reservoir is emptied, there is still fluid remaining in the air vent, and the siphon won't break until air can get into the circuit.

That break takes place, when with all the fluid removed from the reservoir, the column of fluid in the air vent comes down into the reservoir, and air enters after it and rushes up through here, and breaks the siphon.

DR. CHEEVER: Very satisfactory. How long does that cycle take?

DR. MUNRO: It depends on the rate of filling of the bladder, ordinarily from two to three hours. The emptying cycle takes place in perhaps a couple of minutes.

DR. QUINBY: How much intravesical pressure do you work up with it?

DR. MUNRO: That depends on how distended you wish the bladder to be.

DR. QUINBY: How much pressure do you get up?

DR. MUNRO: Pressure up to over 80 centimeters of water, if you raise the siphon sufficiently high. Personally, I never have run over 30 centimeters.

In the case that had the cystitis and tenesmus, where the bladder was very much shut down, and also the case with an automatic bladder, where the bladder was even more shut down, we ran both those bladders on a high intervesical pressure and stretched the bladder out.

The report covering the therapeutic part of this is only preliminary and I know a great many changes will have to be made in our conclusions, but this method has now been used on ten spinal cord injuries at varying times, the longest being a year, and it has proved very satisfactory in our hands. If it has done nothing else, it has simplified the nursing problems in these cases, and I am sure it has reduced the incidence of decubitus ulcerations. We had one case completely free of these ulcerations for four months who was allowed to re-

main wet for twenty-four hours as a result of a mishap to the apparatus, whereupon he promptly developed a bedsore. We healed all the others, I think, easier than we were able to do previous to the use of the apparatus.

In answering Dr. Quinby's questions, I think that the whole problem of bladder recovery and life and death in these cases is so mixed up that an apparatus can't cure one or prevent the other without affecting the first or the second.

There is no question that, as you say, urinary infection is the major factor in the production of death after spinal cord injury, particularly transection of the cord, but there is also no question, I think, that provided the injury to the cord is above the sacral segment, the bladder may reasonably be expected to recover and resume essentially normal activity, if you can keep down the systemic infection that will result from the presence of residual urine or overdistention of the bladder.

Our experience has been that this cannot be done with ordinary indwelling catheter drainage on account of difficulties in actually draining the bladder, due to the position of the inner end of the catheter. Hydraulically speaking, those bladders drain by siphonage. If the bladder is atonic, your siphon won't empty the bladder cavity completely unless the bladder end of the catheter remains constantly under water, and it is almost impossible to arrange, at least in my clinic, to have it stay constantly in that position in the bladder. It is either moved out or pushed in too far, or partly plugged, or, in any event, residual urine seems to collect and with that, infection develops.

I am glad now that there is some reliable information about the regurgitation of urine up the ureters in these spinal cord cases. Dr. Quinby's statement contradicts a statement made by Fullerton, who studied some of Holmes's cases, immediately following spinal injury, with a cystoscope. In those cases he claims he was able to show that the ureteral activity was normal and that there was no giving way of the ureteral valve. He also claims that he did this to these cases during a period of spinal shock. I don't know any confirmatory or non-confirmatory work other than that.

IS THE CONTROL OF TUBERCULOSIS IN SIGHT?

The marked decrease in the mortality from tuberculosis is the outstanding achievement in the modern public health campaign. In 1900, deaths from all forms of tuberculosis in the ten Original Registration States and the District of Columbia numbered 195 in 100,000 population. In 1933 the rate was only 56 in the same area. This is a decrease of 71 per cent since the beginning of the century and represents a saving of 170,000 persons who would have died from tuberculosis in 1933 had the rate of 1900 prevailed throughout the United States in that year.

Beyond question, the general improvement in the environment in which people live and work has favorably influenced the mortality from tuberculosis; but the application of our increased knowledge of this disease and of its treatment has also helped materially to reduce the number of deaths. Add to the saving in mortality the number of cases that have been arrested; the number of persons who have been completely or partially restored to work; the number of young children who have been spared from sickness and orphanhood; and the number of families that have been saved from disruption by the death of a parent from tuberculosis, and we have a most gratifying picture of the splendid achievement of the fight against this disease. The agencies of education and of treatment built up by the anti-tuberculosis campaign have been both efficient and successful.

Even during the trying period of unemployment and of economic unrest, the deathrate from tuberculosis has continued to decline. In 1933, deaths from tuberculosis numbered 59.5 per 100,000 in the United States as a whole, as against 71.0 in 1930, a decline of 16.2 per cent in three years. By every indication the figure for 1934 will continue the trend of improvement.

When the states are considered separately, we find considerable variability, however. The range of the decline in the last three years is between a maximum of 32 per cent and a minimum of 2 per cent. Two states show increases, 9 per cent in Idaho and 3 per cent in Delaware. Seven states show decreases from tuberculosis of 25 per cent or more; 10 a drop ranging between 20 per cent and 25 per cent; 17, between 15 per cent and 20 per cent; 6, between 10 per cent and 15 per cent. In the remainder there were declines of less than 10 per cent. These variations in the recent decline in tuberculosis mortality should, however, be considered in relation to the relative ranking of the states in their tuberculosis deathrates, since, as the deathrates from tuberculosis approach a minimum, it becomes increasingly difficult to effect further reductions. It is, therefore, not to the discredit of states with low tuberculosis mortality rates when they fail to show large declines from year to year. There are some striking exceptions even to this general rule. A few states with very low rates show pronounced decreases since 1930, North Dakota and Utah, for example.—*Bulletin Metropolitan Life Insurance Company.*

DIAPHRAGMATIC HERNIA AT THE ESOPHAGEAL HIATUS, THE SHORT ESOPHAGUS AND THORACIC STOMACH*

BY P. E. TRUESDALE, M.D.†

IN recording the fourth in a series of six cases of diaphragmatic hernia at the esophageal hiatus in adults and involving the stomach alone, we found the attendant problems engaging. The severity of symptoms and the analogy of the complaints with those of angina pectoris were somewhat striking. The appearance of only a small portion of the stomach above the diaphragm, the x-ray manifestation of congenital short esophagus, and a not entirely satisfactory end-result, place this case in a debatable category of herniae of the diaphragm.

The appearance of the herniated portion of the stomach in a breach of this type is not always uniform. Though our group of cases operated upon is too small to warrant dogmatic statements in accounting for variations manifested, nevertheless in every case we have observed evidence of old inflammatory changes in and around the hernia somewhat similar to those found in umbilical hernia and less frequently in inguinal hernia. Adhesions of the stomach occur within the hernial sac. Fibrous bands connecting the sac with the esophagus, pericardium, and lung are common if not characteristic of this type of hernia. No doubt they cause suspension and angulation of the cardia and mechanical interference with the passage of food. Likewise, the associated deformities may make ejection of food from the stomach difficult. In the case recorded below, our attention was called to the appearance of this phenomenon by the patient's physician, Dr. Byron J. Brown, who described the fluoroscopic picture of barium in the lower esophagus as fluctuating like a column of mercury under varying degrees of pressure in a manometer.

CASE REPORT

CASE IV. I. D., female, aged forty-three, housewife, was admitted to the Truesdale Hospital, December 12, 1933. Her only offspring was a premature infant born in 1914. The child died after seven months. The patient had a tonsillectomy in 1905 and hysterectomy in 1915. When admitted her chief complaint was upper abdominal and substernal pain, radiating to the left shoulder and down the left arm. The onset occurred in 1930. While pulling on a frozen clothes-line she felt something tear or give way suddenly in her left upper chest. The following day she felt sharp pains across the extreme upper abdomen. For the next three weeks she was in bed with the same severe pain and vomited frequently. Her attending physician diagnosed the attack as biliary colic and advised her to be operated upon. After she recovered from the attack, she was able to continue her usual household duties. Nevertheless there persisted a feeling of fullness in the epigastrium and pain in the left upper thoracic region after eating, more troublesome when she was in the recumbent position. In 1931 another severe attack of upper abdominal pain was ascribed to gall stones. She was sent to the Rhode Island Hospital where a cholecystogram demonstrated a normally functioning gall bladder and no indication of the presence of stones.

Nine months later in April, 1933, the pain became more persistent. It was epigastric, substernal, radiating to the back and down the left arm to the elbow. At times she would sense a feeling of suffocation, dyspnea, and tachycardia. Further x-ray examination at the Rhode Island Hospital revealed a diaphragmatic hernia. She was given symptomatic treatment.

For the intervening period of eight months this patient has been unable to do her housework. She has lost twenty pounds in weight. Another roentgenologic study of her case was made by Dr. Russell R. Hunt. The existence of a diaphragmatic hernia was confirmed and Dr. Brown referred the patient to our clinic for operation.

Very little evidence of a helpful nature was found on physical examination. The patient was an intelligent, coöperative woman. Her pupils were equal and regular, reacting to light and accommodation. Reflexes were normal. Systolic blood pressure was 130, diastolic 80. Blood vessels were not tortuous or sclerotic. The heart sounds were normal and the lungs clear.

The impression of the examiner was that the diagnosis of diaphragmatic hernia could not be made clinically, that it must be contingent upon roentgen-ray findings.

X-ray examination of the chest on December 7, 1933 showed the lungs clear. The cardiac shadow was normal, but there was a rounded area of decreased density to the left of the spine behind the heart. Its upper level was opposite the eighth interspace posteriorly.

The esophagus appeared normal down to the level of the eighth interspace where it joined the stomach. Its course showed no kinking or redundancy. The hiatus in the diaphragm was opposite the eleventh rib posteriorly and was dilated. The body of the stomach, pylorus, and first portion of the duodenum appeared normal. Impression: congenital short esophagus, diaphragmatic hernia at the *hiatus esophageus*.

Cholecystograms revealed a smoothly outlined, dense gall bladder shadow at fifteen hours and no visible calculi. Films one hour p. c. showed complete disappearance of the shadow.

The electrocardiographic tracing showed myocardial changes as indicated by the slurring of the Q R S waves in all leads and left axis deviation.

DIFFERENTIAL DIAGNOSIS

Dr. William Mason: The symptoms in this case illustrate well the problems involved in the clinical diagnosis of esophageal hernia of the diaphragm. The attacks of pain in the upper abdomen and left chest radiating to the back and down the left arm when accompanied by dyspnea immediately suggest a cardiac origin. The atypical radiation of the pain to the back, its occurrence without relation to exertion, and the limited degree of cardiac pathology would seem sufficient to rule out anginal attacks of coronary disease.

*Read before the Rhode Island Medical Society, March 1, 1934.

†Truesdale, Philemon E.—Surgeon, Truesdale Hospital, Fall River, Mass. For record and address of author see "This Week's Issue," page 271.

Radiation of pain to the back and the presence of gaseous indigestion, distress and vomiting, especially when occurring in a female patient of middle age, are rather characteristic of gall bladder disease. The tendency of the pain to radiate more prominently to the left side does not rule out this diagnosis but is, nevertheless, unusual. The demonstration of a normally functioning gall bladder by x-ray is, however, the strongest evidence against this diagnosis.

If, then, we view the symptoms in relation to the other upper abdominal organs, we do not find them definitive of any disease picture. They have not the regularity or food-relief characteristics of peptic ulcer. There is no confirmatory evidence of carcinoma of the stomach or obstructive lesion in the colon. A pancreatic disturbance cannot be ruled out except by positive evidence establishing another diagnosis. The clinical picture, however, offers little more than a suggestion of pancreatitis.

There is, however, one feature in this case not common to those similar in other respects, namely, the acute onset with the sensation of something giving or tearing in the left chest and occurring coincident with a strain entailing a rise of intra-abdominal pressure. This feature, especially when viewed in the light of subsequent symptoms, suggests strongly diaphragmatic hernia. Finally, extensive traumatic rupture of the body of the diaphragm with migration of the stomach, small intestine, colon, and spleen into the thoracic cavity usually affords physical signs permitting a purely clinical diagnosis; the smaller herniation of a portion of the stomach through the esophageal hiatus gives no such signs, and therefore a clinical diagnosis cannot be expected. The presence of this condition, however, must be suspected in a patient with upper abdominal and left thoracic pain, anginoid in character, when accompanied by digestive symptoms aggravated by lying down, and suggestive but not typical of the more common upper abdominal lesions. In such cases no other diagnosis should be made by the process of exclusion until esophageal hernia has been ruled out by an x-ray examination. This examination can be considered adequate only if the patient has been examined lying on the back in the Trendelenburg position following the ingestion of barium.

OPERATION

On December 12, 1933 under gas-oxygen-ether positive pressure anesthesia administered by Dr. Albert H. Miller, the region of the hernia was exposed through a transthoracic approach. The cardiac portion of the stomach was observed projecting through the esophageal aperture which was considerably enlarged. The stomach covered by a very thin membrane was adherent to the pericardium, the base of the left lung, the pleuroperitoneal margin of the hernial aperture and for several centimeters along the esophagus itself. (See figure 1.) The stomach was easily separated from the esophagus by sponge dissection, then freed from adhesions to the lung and pericardium, and reduced to its normal position below the diaphragm. The phrenic nerve was then oriented, elevated, and injected with alcohol. Closure of the aperture in the diaphragm which was approximately six centimeters at its widest point constituted the next step. At the mesial portion which formed the base of a triangular opening made by the diverging crura, there was some difficulty in obtaining substantial muscular tissue to reduce the hiatus to a size more nearly normal. (See figure 2.) The thoracotomy incision was then closed.

The patient made a good postoperative recovery. Her only complaint was pain and soreness in the primary incision. On January 13, 1934 after a convalescence of uncomplicated progress she was discharged, apparently relieved of symptoms for which operation was done.

This woman entered the hospital for observation August 21, 1934 approximately eight months after operation. Although the symptoms of dysphagia have disappeared and deglutition is now unimpeded

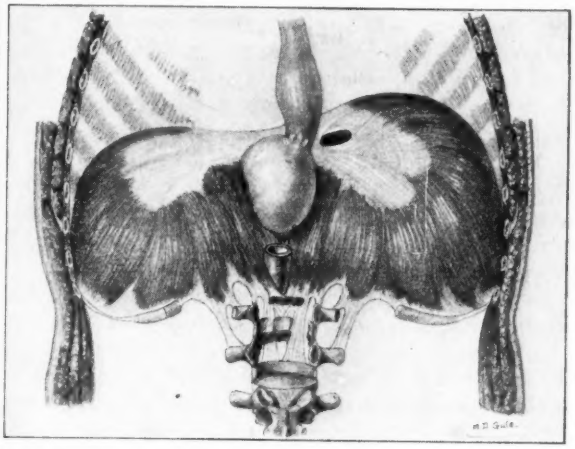

FIG. 1.

and without distress, she still suffers, as before operation, with pain under the left costal margin radiating to the back and at times down the left arm. She also complains of occasional tachycardia. She states, however, that she feels stronger than before operation but has gained only two pounds in weight.

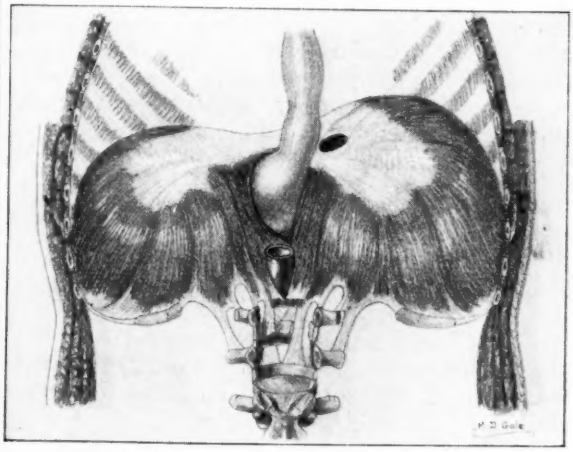

FIG. 2.

We kept her in bed for a week on a Sippy diet. During this time she felt much improved, and continued this regimen after leaving the hospital. Therefore, though this patient was not cured of all the complaints for which she entered the hospital, she has maintained her weight, gained in strength, and is relieved of the disturbing symptoms associated with deglutition.

The x-ray film showed the stomach in the same position as at the time of discharge. The esophageal hiatus was smaller than before operation, but it still appeared to the roentgenologist that a small portion of the cardiac end of the stomach was above the diaphragm. Thus we may consider this patient improved but not entirely well.

OPERATIVE PROCEDURE

We continue to operate for diaphragmatic hernia by a transthoracic approach for several reasons. First, cutting down upon the hernia is a universal practice in operations for inguinal, umbilical, ventral, and other forms of rupture. Secondly, most diaphragmatic herniae develop adhesions and become attached to structures native within the thorax. When the transposed viscera of the abdomen cannot be reduced, it is important and helpful to have the cause of this difficulty under direct vision. This is particularly true in hiatus hernia when one needs to determine whether the obstacle to reduction is a short esophagus, adhesions, ulcer, or malignancy. Thirdly, when the hernial sac is found to be a very definite structure, it can be removed or, as in the second of our cases, obliterated by plication.

The abdominal route is preferred by some surgeons. In favor of this approach it may be said that the broadened esophageal hiatus is more accessible. The crura of the diaphragm which embrace the esophagus cross in front of the aorta on the way from their tendinous attachment to the first, second, and third lumbar vertebrae. The hiatus is immediately in front of the aorta. Its accessibility depends upon the architecture of the individual. When the herniated portion of the stomach returns readily to its position below the diaphragm or can be drawn downward to a sufficient degree to permit lysis of adhesions with safety, the abdominal approach is more simple. The abdominal incision requires less time to make and to close and may be converted into a combined thoracoperitoneal approach if thought desirable.

There was a conflict of opinion in this case as to the existence of a congenital short esophagus. The roentgenologist after inspection of the films and a painstaking fluoroscopic examination reported hernia of the diaphragm associated with congenital short esophagus. The hiatus hernia which was discovered corroborated the diagnosis of Dr. Russell R. Hunt of Providence, R. I. The short esophagus was a new observation. Therefore, we made the lower end of this structure the object of careful scrutiny during the operation. After separating adhesions around and within the hernia and exposing the esophagus, it seemed to the operator that this tube was of normal length. Never having detected a short esophagus, I was liable to error although the structure was in plain sight. The roentgenologist also viewed the esophagus *in situ* and believed it to be shortened.

That two minds after direct inspection could not agree upon the length of the esophagus seemed incredible. Yet this required definition of its lower end in relation to surrounding structures,—the diaphragm, pericardium, vertebrae, and ribs. This topographical relationship ordinarily is not easy to establish during operation. The time element is important. Examination in this case necessarily had to be brief. With the heart, lung, and diaphragm in constant motion, inspection left the observer with little more than an impression. Thus the surgeon saw the esophagus long and the roentgenologist observed it short. The postoperative film showed gastric rugae above the diaphragm, but we know from postmortem examination that there are rugae in the lower portion of the esophagus, not so large as those below the cardia, but nevertheless definite.

So rarely is true thoracic stomach associated with congenital short esophagus reported, that Bailey[1] when writing of the condition in 1919 made the following statement:

> "There have been many cases reported of hernia of the stomach into the thorax but no actual case of development of the stomach within the thorax."

He then reported a case in which the stomach was found lying between the pleural cavities and behind the pericardium in the posterior mediastinum completely surrounded by a serous sac continuous with the peritoneum through the hiatus of the diaphragm. He concluded that there was no evidence that the stomach had herniated through this hiatus. Later we shall return to a discussion of this case in order to present another interpretation based upon failure of fusion of the two halves of the diaphragm instead of failure of the esophagus to elongate.

CONGENITAL SHORT ESOPHAGUS ASSOCIATED WITH THORACIC STOMACH OR PARTIAL THORACIC STOMACH

Our investigation of the literature has revealed the following cases of congenital short esophagus in which a portion or all of the stomach was discovered in the thorax either by x-ray or at operation or autopsy. These cases we have divided into the following two groups:

Cases of this type have been mentioned by Taylor[11], Dunhill[12], and Morison[13]. It is apparent that this type of "thoracic stomach" is fairly common and differs from cases of complete thoracic stomach in which all of the stomach is in the thorax and the pyloric end occupies the esophageal foramen. These cases, twenty in number collected from the literature, are listed in the following chart:

Brun refers to a case reported by Kinney, who said that in his patient the stomach had the position of that of a nine-millimeter embryo. Hitzenberger[33] in a doctor's thesis on the diaphragm published in Vienna in 1927 reports two cases of complete thoracic stomach, but gives no details.

In Group B twelve of the cases were diagnosed at autopsy and eight during x-ray examination. In determining the correct length

of the esophagus, the x-ray film may be misleading, as in the following case of *hiatus hernia vera*. The details leading to death were described and the postmortem specimen exhibited by Dr. Halsey De Wolf and Dr. Bertram E. Clarke on the occasion of this meeting of the Rhode Island Medical Society. In many respects the specimen was unique. The roentgen-ray film was excellent but misleading and the diaphragmatic hernia had no bearing on the cause of death.

GROUP A

CASES OF PARTIAL THORACIC STOMACH

Freud and Horner[2], 1922	M 30	Stomach, ileum, colon, spleen in thorax.	Operation, abdominal route, death.
Hollaender[3], 1928	M 40	Hour-glass stomach, most of it in thorax.	X-ray diagnosis.
Wright[4], 1929	M 7	Widely separated crura of diaphragm; stricture of esophagus.	Dilatation of esophagus; death, perforation by bougie.
Harrington[5], 2 cases, 1930	F 54 M 12	One-half stomach in chest. " " " "	Phrenicotomy, abdominal route, recovery.
Lyons[6], 1930	M 40	Hour-glass stomach.	X-ray diagnosis.
Findlay and Kelly[7], 1931	9 cases 7 males 2 females	Esophageal stenosis, partial thoracic stomach.	X-ray diagnosis.
Monkhouse and Montgomery[8] 1933	7 cases In adults	Stenosis of esophagus, partial thoracic stomach.	Operation advised for non-obstructive cases; x-ray diagnosis.
Clerf and Manges[9], 1934	14 cases 9 females 5 males	Cardiac end of stomach in chest; stenosis of esophagus.	X-ray diagnosis.
Goodall and Hoyt[10], 1934	5 cases 3 females 2 males	Varying portions of stomach in chest.	Ulcer present in some cases; x-ray diagnosis.

GROUP B

CASES OF COMPLETE THORACIC STOMACH

Author	Number	Year	Sex	Age	Diagnosis Made
Bright[14]	1 case	1836	F	19	Autopsy.
Arnheim[15]	1 case	1896	M	32	Autopsy.
Dietlen and Knierim[16]	1 case	1910	F	23	X-ray.
Bund[17]	1 case	1918	M	8 mos.	Autopsy.
Bailey[18]	1 case	1919	M	77	Autopsy.
Huffman[19]	1 case	1920	F	50	Autopsy.
Brun, et al.[20]	1 case	1922	M	11	X-ray.
Hume[21]	1 case	1922	M	56	X-ray.
Plenk[22]	1 case	1922	M	44	Operation and Autopsy.
Tonndorf[23]	1 case	1923	M	76	Autopsy.
Le Wald[24]	2 cases	1924	M	7	Operation.
		1924	F	69	Operation.
Fineman and Conner[25]	1 case	1924	M	22	X-ray.
Roberts[26]	1 case	1927	F	4	X-ray.
Stenzel[27]	1 case	1929	F	Infant	Autopsy.
Morris[28]	1 case	1929	F	44	X-ray.
Koppenstein[29]	1 case	1930	M	75	X-ray.
Jenkinson[30]	1 case	1931	M	68	Operation.
Beutel[31]	1 case	1932	M	30	X-ray.
Jungfer[32]	1 case	1933	M	60	Autopsy.
	20 cases				

CASE REPORT

The patient was a man fifty-eight years old who was admitted on the medical service of the Rhode Island Hospital. He was critically ill with thoracic and abdominal symptoms. His respirations were rapid and labored. He had abdominal pain, distention, and vomiting.

An x-ray film of the esophagus and stomach after a barium meal taken at a previous admission nine months earlier revealed what appeared to be a typical case of thoracic stomach with short esophagus. The point at which the barium column defined the lower end of the esophagus was on a level with the

body of the seventh dorsal vertebra. Three days after admission the patient died. Autopsy revealed as the cause of death an acute intestinal obstruction from a strangulated inguinal hernia reduced *en bloc*. As an associated anomaly there was a hiatus hernia of the diaphragm. The hernial sac and its contents consisting of the entire stomach and omentum occupied a paramedial position in the right thoracic cage. The autopsy confirmed the x-ray findings.

Through the courtesy of Dr. Clarke, who during the autopsy had removed the diaphragm with all

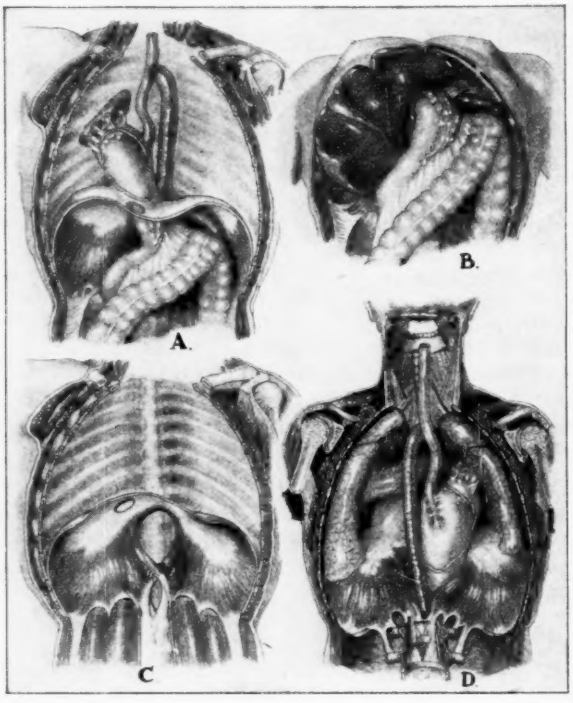

FIG. 3.

adjacent structures, we obtained a loan of the specimen for study. On inspection of the gross specimen we were convinced that the esophagus was several inches short. (See figure 3A.) A view of the diaphragm from the under side showed the pyloric end of the stomach protruding from the esophageal hiatus. (Figure 3B.) When the stomach was drawn down through the aperture, a greatly enlarged esophageal hiatus was revealed. (Figure 3C.)

Upon minute dissection of the structures involved in the hernia, however, we found that the esophagus, instead of terminating at the point indicated in the roentgen-ray film, continued downward enveloped in adhesions and contiguous with the stomach wall, for a distance of about two and one-half inches before it reached the cardia. (See Figure 3D.)

When we returned the specimen to Dr. Clarke, we sought his assistance in checking our findings. His communication reads as follows:

"I have sectioned that portion of the esophagus which you discovered in our diaphragmatic hernia and find that you are right. That entire portion is lined by stratified squamous epithelium. This adds about two inches to the apparent length of the esophagus."

Therefore, allowing an inch for the slack in the upper esophagus as observed in the film, we are in all likelihood justified in concluding that the esophagus which in this case was apparently shortened by three inches or more, in reality was of normal or nearly normal length.

In approaching the subject of thoracic stomach one is faced with the question, "Is the stomach in the thorax because the esophagus is short, or is the esophagus short because the stomach is in the thorax?"

Opinions differ on the developmental order of this phenomenon. Those who say that the congenital foreshortening of the esophagus prevented the descent of the stomach from the thorax, where it normally develops, into the abdominal cavity, where it is normally found are Dietlen and Knierim, Bund, Bailey, Hume, Plenk, Tonndorf, Le Wald, Hitzenberger, and Roberts. Barrett and Wheaton[34] quote Keith as follows:

"The stomach is originally almost cervical in position, but as the pleural diverticula expand and the diaphragm descends, the esophagus becomes elongated and thus the stomach also descends maintaining its position below the diaphragm. If the esophagus does not elongate, the descent of the stomach is arrested and thus we find it placed within the posterior mediastinum surrounded by its sac of peritoneum. The sac is not formed by the protrusion of the organ but by the downward movement of the diaphragm over the stomach which is stationary."

Thus we have eminent opinion that failure of the esophagus to elongate occurs in the early embryo when orderly changes of position are taking place by other structures in this region. Several authors, however, are reluctant to subscribe to this view. Huffman and Jungfer believe that at birth the stomach was in the abdomen, having descended normally in the embryo, and later, either at birth or shortly afterwards, it was pulled up by the short esophagus through a weak spot in the diaphragm at the esophageal hiatus. The hiatus was weak congenitally because the stomach developed more cephalad than usual and required more space in the esophageal opening than would be needed by the esophagus alone.

Arnheim, Stenzel, Beutel, and Koppenstein are unable to decide whether the shortness of the esophagus is the *cause* of the position of the stomach above the diaphragm, or the *result* of failure of organs to descend into the abdomen before birth.

Jenkinson declares, "The esophagus is usually short, owing to the high position of the stomach."

Bright and Morris offer no hypothesis. Fineman and Conner do not discuss the embryological process, but merely state that on account of the short esophagus, this type of hernia is irreducible.

Since cases of thoracic stomach and short esophagus are multiplying in the literature, the time is now propitious for an accounting. There is need to return to the embryological founda-

tion for basic observations. To interpret the embryological shuffle correctly, more than a surface knowledge of the subject is required. Like Sir David Brewster's kaleidoscope the fragments are so arranged that changes of position exhibit an endless variety of symmetrical forms. An asymmetrical fragment may spoil the instrument. So it is in the developmental activity of the embryonic diaphragm. At this point it is important to heed the frank admission of teachers of embryology that comparatively little pertaining to the shifting of the diaphragm and its adjacent structures in the very young embryo is either known or understood. With our present knowledge, clinical and postmortem evidence is hard to interpret. Supposition is inevitable. A theory which seems plausible today may be discarded to-morrow. Since the evidence is not yet entirely conclusive, we venture to account for thoracic stomach and short esophagus in terms that vary from those already expressed.

EMBRYOLOGY

It is apparent that originally the anterior margin of the embryonic disc is bent ventrally by a fold of the germ layers. As the form of the body straightens out, those structures which are in the cephalic and cervical regions recede caudally to the thorax and abdomen.

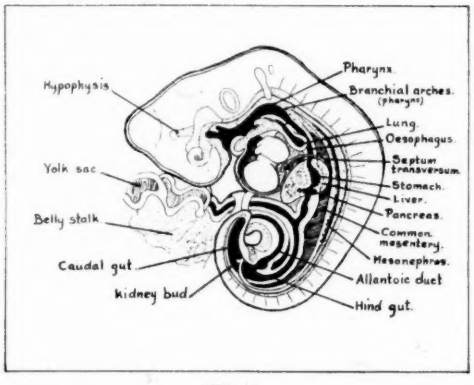

FIG. 4.

Figure 4, an illustration of a human embryo of five millimeters from the work of Bailey and Miller[35], offers a good demonstration of the position and relations of the esophagus, diaphragm, and stomach when the primitive gut becomes differentiated into distinct regions. The esophageal region forms a comparatively short tube of uniform diameter extending from the pharynx of the stomach. The diaphragm at first has a broad attachment to the cephalic surface of the liver. It is essential to note also that *in this very early embryo the stomach is below the diaphragm.* The liver is the largest of the abdominal viscera and is next to the brain in size. It grows rapidly. Its broad attachment to the diaphragm is gradually separated but not entirely. At this time the mesenteries are of importance in showing the attachment of the stomach to the liver. The ventral mesentery, which is directly continuous with the septum transversum, becomes attached to the liver and the lesser curvature of the stomach. It also extends along the duodenal portion of the gut for a short distance.

Thus far, then, we have in the second week of fetal development the diaphragm attached to the esophagus, the cardiac end of the stomach, and the upper surface of the liver. The lesser curvature of the stomach and the first portion of the duodenum are attached to the under surface of the liver by the gastrohepatic ligament. As these structures migrate caudally, changes in position and structure are very complicated. In their movements the diaphragm and liver lengthen the esophagus, and by their attachments not only draw the stomach down, but rotate it so that ultimately a large part of the lesser curvature lies almost horizontal with the under surface of the liver. When caudal migration proceeds, their interlocking relationship is adequate evidence that the esophagus elongates and that the stomach and liver maintain their relationship below the diaphragm.

In the chapter on *Teratogenesis* in Bailey and Miller's work on *Embryology*[36] we find reference to defects in the thoracic and abdominal regions as follows:

> "The digestive tube and ventral body wall are formed primarily by a bending ventrally and fusing of the originally flat germ layers. The splanchnopleure on each side first bends ventrally and fuses with its fellow of the opposite side in the medial line to form the gut, and soon afterward the somatopleure likewise fuses in the ventro-medial line to form the body wall. Naturally a defective fusion of the two sides of the somatopleure would result in a more or less extensive medial cleft. The cleft may be limited to a small portion of the abdomen or thorax, or may extend from the neck to the pelvis."

If there is incomplete fusion medially between the two halves of the diaphragm, a cleft of varying dimensions remains between the crura. The esophageal hiatus, which is the space between the crura, varies greatly within normal limitations. We make digital examination of the esophageal hiatus a routine procedure in upper abdominal operations. From actual observations we have found that normally the hiatus will admit the tips of two fingers. Sometimes there is room for the index finger only; on other occasions we have had no difficulty in passing three fingers into the hiatus. Whether the latter is normal or abnormal, we do not know. Perhaps an aperture of such size represents a site of potential hernia. Brown-Kelly[37] has demonstrated that temporary hiatal hernia in children is not rare. He considers it a physiological variation.

If fusion at the hiatus has been incomplete in the embryo, and if a loose attachment at the lower end of the esophagus exists, no transposition of the stomach is likely to take place *before birth*. As soon as the infant is born, however, new mechanical factors are introduced. Respiration is established and the diaphragm begins to function. This results in producing a negative pressure in the thorax and a variable positive pressure in the abdomen. Thus, when the baby cries, the cardiac portion of the stomach is forced upward into the breach, if one exists. Aided by suction, a loose attachment of the terminal esophagus, and a sizable cleft, it is conceivable that a large portion of the

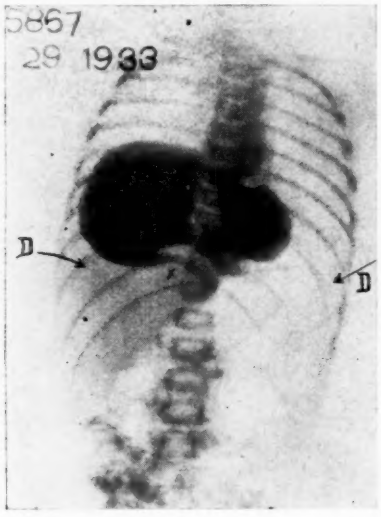

FIG. 5.

stomach or the entire structure may pass promptly into the thorax and remain there, gradually making adjustment of position with other organs. This transposition of the stomach may exist for many years or a lifetime without causing symptoms because of the early adjustment of the organs in the thorax. An illustration of this transposition of the stomach in early life may be seen in the following case.

I learned of this case through a personal communication from Dr. Peterson of Roanoke, Virginia, who x-rayed a male infant admitted on the service of Dr. Hugh Trout at the Jefferson Hospital. The baby had several gastric hemorrhages but was apparently perfectly normal between attacks. There was loss of weight and some degree of acidosis. X-ray examination revealed an esophagus which pursued an irregular course and the cardiac end of the stomach above the dome of the left diaphragm. (See figure 5.) The barium crossed the spine and appeared as a large mass above the liver in the lower right chest, then recrossed the midline at the point X in the illustration and from this point progressed rapidly into the duodenum. The filling defect over the spine is secondary to pressure. The letter D indicates the level of the diaphragm. Dr. Peterson and Dr. Moore of Washington interpreted this as a case of thoracic stomach. The baby was not operated upon and was discharged from the hospital improved after medical treatment.

The fact that the esophagus pursued a sinuous course indicates that it was apparently of normal length. Moreover, the history of several gastric hemorrhages since birth suggests hernia of the dia-

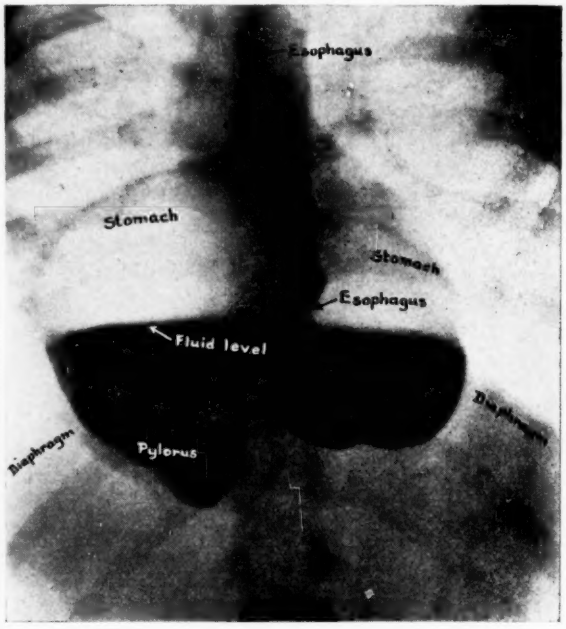

FIG. 6.

phragm rather than thoracic stomach. It is highly probable that the stomach was in the abdomen at birth and, as in the other cases discussed above, when the baby coughed or cried, was thrust upward through a developmental defect in the diaphragm and remained incarcerated in the chest.

According to this theory, cases of complete thoracic stomach as in Group B demonstrate a continuation of the process of herniation begun in cases of partial thoracic stomach as in Group A. If the entire stomach is transposed soon after birth and the pylorus only left in the hiatus, this opening may gradually close to a degree which might make it appear that the stomach had never been below the diaphragm. The stomach would then assume a position above the diaphragm, and the congenitally enlarged hiatus would tend to close during the early months of life, preventing any possible re-entrance of the stomach into the abdomen. A case which came under the observation of Roberts[38] of Liverpool illustrates this condition of thoracic stomach. Roberts believes that the stomach developed congenitally in the thorax.

CASE REPORT

E. W., aged three years and ten months, the only child of normal parents, undersized and constipated, was admitted to the Liverpool Hospital for x-ray examination. At the age of one year she began to eject food and vomited thick dark-brown mucus. At the age of two, she vomited after each meal and became dyspneic after eating.

X-ray examination showed the entire stomach in the posterior mediastinum. (Figure 5.) The esophagus showed no obstruction. The stomach was bilocular, and both cardiac and pyloric portions presented a rounded dome containing air above the fluid contents. The pylorus occupied an opening in the right leaf of the diaphragm just below the eleventh rib.

The patient was kept under observation and when last seen in 1932 was leading a normal life as an alert child of ten years of age. She still held to a strict diet avoiding foods which would cause symptoms. The position of the stomach at this time as revealed by x-ray is shown in figure 6.

This case admits of two possibilities. The stomach may have developed in the thorax, but it is plausible to suppose that the stomach descended to its normal position below the diaphragm and later was forced through a hiatus in the right leaf of the diaphragm which resulted from failure of fusion embryologically. From the x-ray film the cardiac end of the stomach appears to be at the level of the ninth costal cartilage and the esophagus of normal length. Therefore the embryo stomach descended below the diaphragm, for probably in no other way could the esophagus have attained normal length. At birth or later, an unusual degree of violent intraäbdominal pressure from one of many causes could have forced the stomach through the congenital aperture in the diaphragm. Once in the thorax, the stomach became trapped and the hiatus gradually contracted thus preventing any further descent of the stomach.

This tendency on the part of the hiatus to close is analogous to closure which takes place in other forms of hernia. In many infants conditions at the internal and external ring predispose to hernia, but go undetected. For those discovered, simple treatment suffices. If the hernia is kept reduced, the hernial ring gradually closes to a point where admission of the bowel to the inguinal canal is blocked, at least temporarily. Nevertheless, a predisposition to hernia remains. In adult life many of these individuals develop "congenital" hernia.

So it is in diaphragmatic hernia. The stage is probably set early for the development of diaphragmatic hernia at the esophageal hiatus. In the embryo, failure of the two sides of the diaphragm to fuse may be of any degree. Separation of the crura may be so marked that hernia develops early. On the other hand, it may be so slight that hernia does not appear until late in life when there is sufficient loss of tone in the structures supporting the hiatus. The stomach, thrust upward from sudden increase of intraäbdominal pressure, rises and in whole or in part assumes an abnormal position in the mediastinal space.

CONGENITAL SHORT ESOPHAGUS

The amount of material reported is still not adequate to provide conclusive evidence upon the incidence of short esophagus. We have operated upon twenty cases of diaphragmatic hernia. Nine have been in children under the age of twelve, five in the middle-aged, and six in late adult life. In addition, we have made postmortem examinations on three infants with diaphragmatic hernia. In this series of twenty-three cases, we have not observed, or perhaps it would be better to state, recognized a single case of short esophagus. Yet we have found numerous reports in the literature. It is well to note, however, that only a small portion of these were confirmed at autopsy.

Carey[39] studied the development of the esophagus in the human embryo by selecting for comparative study corresponding portions from the esophagus in an ascending series of embryos. He discovered that in embryos from nine to fourteen millimeters the esophageal epithelial tube grows relatively more rapidly in width than in length. Later there is a much more rapid development in length. This elongation is due to the descent of the stomach and to the resistance to diametrical growth presented by the inner close spiral musculature. The epithelial tube pursues the line of least resistance and elongates in response to traction. If we compare a fourteen-millimeter embryo with a twenty-four millimeter embryo, we find that the length of the esophagus has quadrupled, whereas the width has merely doubled. Therefore, Carey states, that if the stomach, through failure to migrate caudally, remains higher than normal, there is less tension on the esophagus which remains short.

This is also true if the stomach, after birth, rises into the thorax, especially in those cases in which there is persistence of the infracardiac bursa and failure of attachment of the esophagus to the diaphragm at the esophageal hiatus. Naturally the esophagus will shorten somewhat if the stomach remains free indefinitely in the thorax.

On the basis of our hypothesis which predicates a congenital failure in fusion of the two halves of the diaphragm at the *hiatus esophageus*, thus allowing the stomach to ascend into the thorax and permitting the esophagus to remain infantile in length through lack of traction, we wish to suggest a different interpretation of the findings at autopsy from those offered by Bailey, Akerlund[40], Tonndorf, Hume and others. The meticulous care with which Bailey conducted his examination and reported the autopsy findings classifies his record as unique. The length of the esophagus was infantile (figure 7) and unlike those in figures 8 and 9 reported by Huffman and Hume, in which the esophagus was of normal or nearly normal length. The evidence adduced is sufficient to convince anyone that the reporter has presented a true thoracic stomach and a developmental short esophagus. However suggestive, this does not

necessarily signify that the esophagus was, *per se*, congenitally short, or that the stomach developed in the mediastinum before birth. Bailey explained the existence of the sac by supposing that the anlage of the stomach rested abnormally far anterior on the alimentary canal

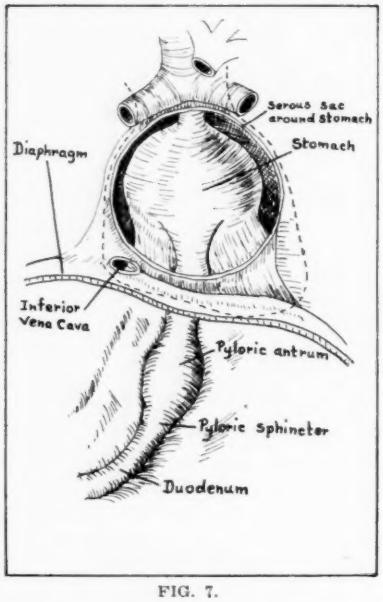

FIG. 7.

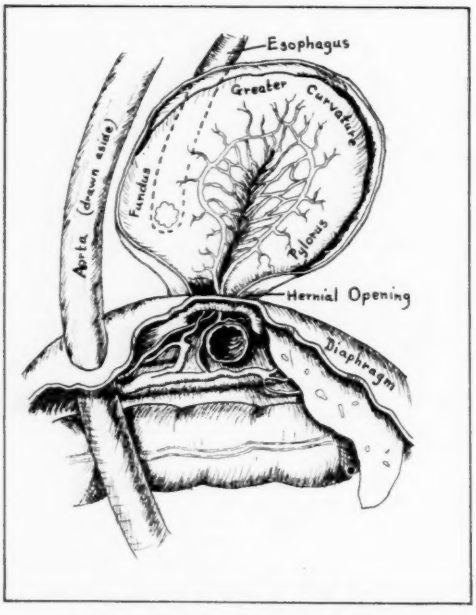

FIG. 8. Huffman's Case—Ref. 19.

and that in the embryo the peritoneal pockets of the stomach developed before stomach and diaphragm began their descent. When the diaphragm descended, it left behind the stomach which was anchored by the esophagus, and from necessity elongated these peritoneal pockets which remained continuous with the peritoneal cavity.

This theory resembles Tonndorf's in assuming that the esophagus is short because of a cessation of growth in very early embryonic life. The septum transversum continues its descent under pressure from the growing thoracic cavity. Since its connective tissue is attached to the esophagus, the shortened esophagus pulls a portion of the diaphragm upward forming a cone-shaped sac, which partitions off the stomach anlage. When later muscularization of the diaphragm takes place, the esophageal hiatus is formed not at the end of the esophagus, but

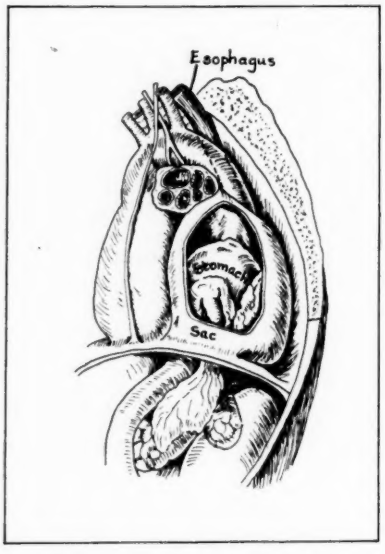

FIG. 9. Hume's Case—Ref. 21.

below at the final level of the diaphragm. By this process the stomach becomes incarcerated in the hernial sac above the diaphragm.

Akerlund states that the esophagus ceases to elongate because of a persistence of a left and a right pneumoentericus recess resulting in complete lack of fixation of the esophagus and no traction upon it. Bund finds a short esophagus possible if the right infracardiac bursa remains patent, allowing complete motility of the esophagus on one side. Plenk agrees, but points out that this explanation holds only for those cases in which the esophageal hiatus is abnormally wide.

Tonndorf refutes the theory that the hiatus is enlarged by failure of the infracardiac bursa to close. This bursa is the anterior part of the *bursa omentalis*, and therefore the hernia sac would be behind the stomach; in all the cases reported the sac is on the ventral side of the stomach.

We are not convinced that the position of the anlage of the stomach or that of the peritoneal pockets was an important factor in the subsequent transition of the stomach. The common embryonic defect which we observe in thorax and abdomen is a median cleft from failure of fusion. Let us assume that such an abnormality existed in the Bailey case. Up to the time of

birth the stomach would have descended in its normal relations with esophagus, diaphragm, and liver. The only abnormality would be a median cleft resulting in an enlarged esophageal hiatus. Pressure, if any, above and below the diaphragm in the fetus is unimportant because it is equally distributed by the amniotic fluid.

Immediately at birth the baby cries and respiration is established. There is thus inaugurated a slightly positive pressure within the abdomen and a negative pressure within the thorax. If the esophageal hiatus is large enough, the entire stomach may be carried into the mediastinum by this combination of negative pressure above and positive pressure below the diaphragm. Once transposed, in rare instances the stomach may remain above the diaphragm. With its ascent there is a slack in the esophagus. This disappears with the growth of the individual, whereupon the esophagus, deprived of the necessity of becoming longer, remains infantile in length. At this period of development when mediastinal structures yield position readily, the stomach establishes itself permanently behind the pericardium without encumbering or endangering life.

The stomach, when in its normal position, is pulled upon by omentum, transverse colon, and liver, and in turn pulls the esophagus downward. Not so, however, when the stomach is forced through the enlarged hiatus at or soon after birth. The *pars pylorica* may be at the hiatus, but the body of the stomach is found above the diaphragm, and when filled or partially filled with food, may actually rest on the diaphragm. As time passes the hiatus closes to some extent, assuring the stomach a permanent place within the thorax. There is no pull on the esophagus throughout life, and it remains the same length as it was at birth. There is no reason for it to become longer.

This sort of peaceful makeshift does not obtain when hernia occurs in adult life. The mediastinum is then more rigid. There is no room to spare. Each structure has already established its position and cannot be encroached upon without protest. Thus, if only a small portion of the stomach passes upward, there occurs some degree of mechanical interference with the passage of food and very often inflammatory changes at the lower end of the esophagus. These mechanical readjustments cannot be made without causing gastric disturbances which are out of all proportion to the size of the hernia. At times symptoms are so distressing that one patient stated that she looked upon mealtime as a "curse".

Huffman's case (figure 8) and one reported by us (figure 3D) fall into another category in which transposition of the stomach occurred at or near the period of life when these individuals had attained adolescence. We arrive at this conclusion from the length of the esophagus as revealed at autopsy.

It is more than a striking coincidence that these two specimens bear such marked resemblance. In each the entire stomach, except the *pars pylorica,* is above the diaphragm. The ventral mesogastrium of the embryo which becomes the gastrohepatic ligament and extends over a portion of the duodenum in each case evidently appeared sufficient to stabilize the position of pylorus and duodenum. In each case the stomach and a portion of omentum were contained in a sac lined by a serous membrane continuous with the peritoneum. The esophagus in one case as in the other approximated its normal length. Instead of ending in the summit of the hernial sac, as in Bailey's case, it continued downward posteriorly to join the stomach only a relatively short distance above the diaphragm.

Huffman's case (figure 8) was that of a woman fifty years old who died a few hours after admission to the hospital. At necropsy there was nothing noteworthy about the diaphragm except the enlarged esophageal opening. In addition to the diaphragmatic hernia the patient had a right labial and a left interstitial hernia. There were only eleven thoracic vertebrae and eleven ribs. The foramen ovale was patent. These minute details attest to the thoroughness of the postmortem dissection. Huffman concludes that this is a case of true diaphragmatic hernia occurring through a congenitally weak esophageal opening, possibly because the anlage of the stomach, as suggested by Bailey, developed more cephalad. Bailey's theory is by no means untenable, but as a basic causal factor the congenital cleft is more impressive than the altered position of the anlage of the stomach which can neither be proved nor disproved.

SUMMARY

1. The fourth case of hiatus hernia of the diaphragm treated surgically by thoracic approach has resulted in partial relief of symptoms and cannot be termed a cure.

2. Research study of congenital short esophagus and thoracic stomach shows many conflicting theories as to origin.

3. A comparative study of our specimens with illustrations in the works of other investigators prompts us to offer a new theory as to the origin of congenital short esophagus and thoracic stomach, namely,

a. The enlarged esophageal hiatus or any other congenital aperture in the diaphragm occurs as a result of embryological failure of fusion.

b. Almost invariably, thoracic stomach is the result of herniation through a congenital hiatus hernia and may occur at any time after birth.

c. The short esophagus is the result of cessation of traction upon it by the stomach which has assumed a position in the thorax.

4. In many instances a short esophagus of a few centimeters is the result of inflammatory changes at its lower end.

5. Reasonably accurate deductions as to the position of the stomach and the length of the esophagus can be made only by careful observation at the operating table or at autopsy.

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STUDIES ON OVARIAN DYSFUNCTION*

II. The Application of the "Hormonal Measuring Sticks" to the Sorting Out and to the Treatment of the Various Types of Amenorrhoea

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IN the first paper of this series[1] values obtained with two hormonal "measuring sticks" on normal individuals were given. One of the most useful clinical applications of these measuring sticks is in the sorting out of the various types of amenorrhoea, especially since the choice of treatment depends on the type of amenorrhoea. A further use may be in the estimation of the success of the treatment instituted.

Since amenorrhoea is the lack of menstruation, a short discussion of the hormonal aspects of this phenomenon is paramount. Our most accurate knowledge in this field concerns the factors influencing uterine bleeding in Rhesus monkeys. As far as is known, only two hormones act on the uterine endometrium, namely the two ovarian hormones, estrin and progestin. The former hormone causes the uterine glands to grow longer (mitoses in epithelium); the latter causes them to secrete (secretory activity in cytoplasm of epithelial cells). Progestin, how-

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ever, acts only after the endometrium has been "primed" with estrin. Since these hormones are apparently the only two involved and since both can now be obtained in active extracts, it should be possible in castrated monkeys to learn just what are the factors associated with uterine bleeding. Such experiments have been reported by Engle, Smith and Shelesnyak[2] and the results seem quite clear. If a female monkey is castrated, the endometrium, of course, becomes atrophied; if now estrin is given for a period of time, this endometrium proliferates; if estrin is now stopped, a regression occurs associated with uterine bleeding. Thus this bleeding is a manifestation of the breaking down of a built-up structure when the substance producing this structure is removed. This is not all, however. If the castrated monkey receives estrin for a period of time and then receives progestin in addition, the endometrium undergoes a further change to the secreting phase; if now the progestin injections are stopped, while estrin is continued, there is again a regression and uterine bleeding occurs. In this case a more complicated structure has been built up with the use of a second hormone and the bleeding is again the manifestation of the breaking down of this built-up structure when the second hormone is stopped. Uterine bleeding in the monkey, therefore, is apparently due to cessation of estrin production in a cycle where only estrin is being produced or to cessation of progestin production in a cycle where both are being produced.

If the factors are as simple as the above experiments indicate, it would be easy to list the possible causes of lack of uterine bleeding in monkeys. The two main headings would comprise those where no endometrial structure was built up because of a lack of hormones and those where the structure, once built up, was maintained because of a persistence of hormones. There would be in addition those where the hormones were all right, but where the structure acted upon was at fault, e.g., some uterine abnormality. The hypo-hormonal group could be further subdivided into primary ovarian lack of function and that secondary to pituitary hypofunction.

These would constitute five groups as follows:

I. Hypoestrinism — primary ovarian.	Hypo-hormonal.
II. Hypoestrinism — secondary to pituitary hypofunction.	
III. Persistent estrin formation, where no progestin had been formed.	Persistent-hormonal.
IV. Persistent estrin and progestin formation.	
V. Endometrial abnormalities.	Normal-hormonal.

The next question is how closely uterine bleeding in monkeys is analogous to menstruation in women. Probably very closely. The most controversial question is whether the anovulatory cycle which occurs in monkeys occurs in women. Since a corpus luteum, and hence progestin formation, is the sequel of ovulation, this question in terms of hormones comes down to whether menstruation is always the breaking down of an endometrium which has been built up by both hormones or whether in some instances it may not be the breaking down of an endometrium which has only been acted upon by estrin. The weight of opinion is swinging toward the latter view. It has been shown by several investigators[3, 4, 5] that oöphorectomized women, like monkeys, have uterine bleeding resembling menstruation about two days after cessation of estrin injections. This has likewise been our experience. Therefore, it is almost fair to say that if an anovulatory cycle did exist in women, it would still be followed by something very similar, if not indistinguishable symptomatically, from menstruation. Therefore the five groups of lack of uterine bleeding in monkeys can probably be used for amenorrhoea in human beings.

I. HYPOESTRINISM—DUE TO PRIMARY OVARIAN HYPOFUNCTION

There are several conditions which cause amenorrhoea of this nature. The most clear cut ones are, of course, bilateral oöphorectomy (see patient 1) and roentgen irradiation of the ovaries (see patient 2). Menopause is a physiological form of primary ovarian hypofunction. It will be discussed more thoroughly in the next paper of this series. Those of the secondary amenorrhoeas which are the menopause coming at an abnormally early age, menopause praecox as it were, also belong here (see patient 3). Infections can destroy the ovary completely enough to cause this type of amenorrhoea. In addition some cases of primary amenorrhoea prove to belong in this group (see patient 4).

The hormonal "measuring sticks" clearly separate this group from all others. The assay of estrin in the twenty-four hour urine ("E")* is, of course, negative. The prolan A test ("P A")* on the first morning specimen is positive, showing that more than normal amounts of this substance are being produced. This important contribution was first made by Zondek[6] and fits in with several other facts, notably that there is an hypertrophy of the anterior pituitary in gonadectomized animals[7] and an increase in their content of prolan A[8]. The myometrium is of course atrophied in this group as estrin is required to maintain it. Figure 1 (q.v.) shows in a schematic way the hormonal abnormalities in this group as compared with the normal.

*By "E" and "P A" we mean the estrin and prolan A test as done in this laboratory[1].

In the first paper of the series[1] dealing with values on normal individuals it was pointed out that these two tests as done at the Massachusetts General Hospital result in E being positive in about 80 per cent of the tests and P A being negative with only a very rare exception.

Patient 1. Hypoestrinism—Primary Ovarian—Bilateral Oöphorectomy.

This patient, aged thirty-one, had her second ovary removed on July 19, 1933 for dermoid cyst, the other ovary having already been removed at a previous operation. Hot flashes began late in August. The hormone tests and their time relationship to these events were as follows:

7-19-33	Operation.	
7-30-33	E = 0	P A = 0
8-7-33		P A = 0
8-14-33		P A = 0
8-20-33	Hot flashes begin.	
9-10-33		P A = +
9-26-33		P A = +
12-11-33	E = 0	P A = 0
12-18-33	E = 0	P A = +
12-26-33	E = 0	P A = +
1-3-34	E = 0	P A = +
1-7-34	E = 14 rat units	P A = +
1-16-34	E = 0	P A = +
1-22-34	E = 0	P A = +
1-30-34	E = 0	P A = +

Comment:—Note the latent period in the establishment of a positive P A. E, of course, becomes negative at once. The relation between symptoms and excess production of prolan A will be discussed in the next paper.

Patient 2. Hypoestrinism—Primary Ovarian—X-ray Irradiation of Ovaries.

This patient, aged forty-one, had her right breast removed for cancer on November 3, 1933. X-ray treatment of the ovaries was started on November 17. The time relationship of the important events was as follows:

11-3-33	Right mastectomy	
11-5-33	Onset of catamenia	
11-17-33	X-ray treatment started	
11-20-33		P A = 0
11-27-33	E = 39 rat units	P A = 0
12-4-33	E = 0	P A = 0
12-11-33	Catamenia begins	

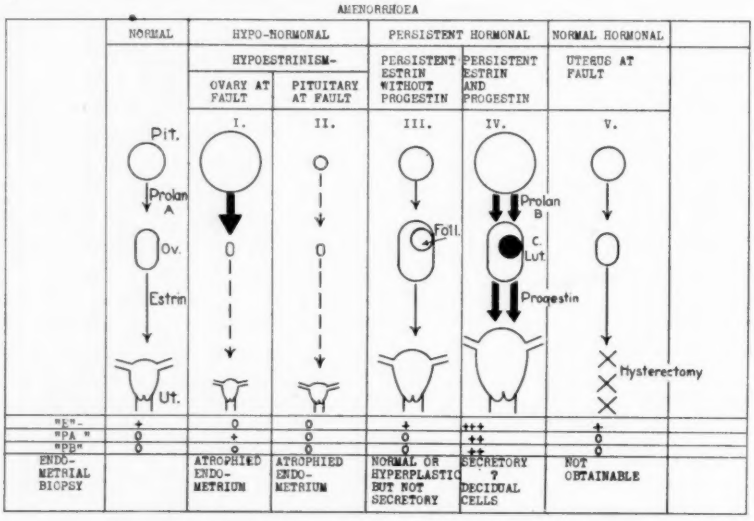

FIGURE 1. Schematic representation of the hormonal abnormalities in the five types of amenorrhoea as compared with the normal, with a chart of the results to be expected in each case with the measuring sticks.

Comment:—Note latent period again in establishment of positive P A. Positive E test on 1-7-34 shows that some functioning ovarian tissue was still present, capable of responding to the excess of prolan A.

Patient 3. Hypoestrinism—Primary Ovarian—Menopause Praecox.

This patient, aged twenty-nine, began to menstruate at twelve; was regular every twenty-eight days until the age of eighteen; she then began to skip periods. She was married six years ago and has had only one baby, though she has wanted more. She gained forty-five pounds in four years. Her periods ceased entirely eighteen months ago. She has had occasional hot flashes. Pelvic examination was negative except for slightly atrophied mucous membranes. Her tests were as follows:

1-3-34	E = 0	P A = +
1-24-34	E = 0	P A = +
2-5-34	E = 0	P A = +

An endometrial biopsy was attempted but no specimen could be obtained, showing that the endometrium was atrophied.

Comment:—Until the hormone tests repeatedly showed that there was an overactivity of the pituitary the clinical impression, because of the obesity and general appearance, was that her trouble was a pituitary deficiency. The tests are similar to those at the menopause and make the diagnosis of menopause praecox almost certain. The hot flashes, however, in themselves should have suggested a primary ovarian hypofunction (v. infra).

Patient 4. Hypoestrinism—Primary Ovarian—Patient with Primary Amenorrhoea.

This patient, a stenographer, aged thirty-one, never had any periods nor any periodic molimina. On physical examination her skin was soft and elastic. She showed almost complete absence of secondary sex development. The axillary and pubic hair was slight in amount. The mucous membrane of the vagina was pinkish in contrast to the normal purplish hue. This is an indication in itself of hypoestrinism. By rectal examination the uterus was very small, but palpable. The ovaries were not felt. Her span was ten cm. greater than her height, which suggests delayed union of the epiphyses and is a manifestation of hypoestrinism, but does not indicate the type. Her tests showed E = O, P A = +.

Comment:—Without the tests it was obvious that this patient lacked estrin. It was impossible to say, however, whether this was a primary ovarian trouble or one secondary to pituitary hypofunction. Primary ovarian deficiency as a cause of primary amenorrhoea is not associated with hot flashes for some reason, so the presence or absence of these is not a distinguishing point. The tests made it clear that the ovary was at fault. The implication as regards treatment will be discussed below.

II. HYPOESTRINISM—SECONDARY TO PITUITARY HYPOFUNCTION

The most obvious example of this group occurs in the pituitary dwarfs with primary amenorrhoea. Such individuals, of course, lack the growth hormone, and usually the thyrotropic hormone, as well as the gonad stimulating hormone. They might be said to have a panhypopituitarism. There are cases in this group, however, where apparently only the gonad stimulating hormone of the anterior pituitary is lacking (see patient 5). Pituitary hypofunction is, furthermore, not uncommonly a cause of secondary amenorrhoea. That large group of individuals who gain a lot of weight and whose intervals become longer and longer and whose periods become scantier and scantier and finally cease probably belong to this group (see patient 6). Patient 7 (q. v.), however, is a patient who developed this type of amenorrhoea out of a clear sky, as it were. In her case the other functions of the pituitary were not affected. Those patients, whose entire anterior pituitary gland is destroyed, develop this type of amenorrhoea as part of the disease complex, Simmond's Cachexia (see patient 8). Acromegalics, of course, often develop an amenorrhoea of this type. Pregnancy with its concomitant functional change in the anterior pituitary may be followed by a state where the pituitary is hypofunctioning (see patient 9). Patients who develop amenorrhoea due to gross dietary insufficiencies likewise fall into this group (patient 10). Finally, the amenorrhoea, associated with debilitating diseases such as pulmonary tuberculosis probably belong here. This type, of course, may be dietary in some instances.

There is one marked difference in symptomatology between patients in group I and group II. Secondary amenorrhoea of the former variety is associated with hot flashes and other symptoms of the menopause; not so with that of the latter variety or only to a minimal degree. Primary amenorrhoea, however, regardless of the type, does not have hot flashes as one of its symptoms.

The hormone tests in this group (see figure I) show P A to be negative and E to be negative. It will be noted that, whereas both tests differed from the normal in group I, here only E does. Furthermore, since E is only positive about 80 per cent of the time in normal individuals[1], it would take several negative tests to be sure that an abnormality was present. One infers that, since there is too little estrin and since prolan A is not increased, it must be decreased. It would be very desirable, however, if it could be shown as well that less prolan A than normal was being excreted. This would require a more delicate test because the method now employed fails to detect this hormone in normal individuals. It is for this reason that an attempt is under way in this laboratory to make use of a method for measuring prolan A whereby the small normal amounts give a positive test[9]. It should then be possible to determine directly rather than by inference that the anterior pituitary is hypofunctioning as regards prolan A.

Patient 5. Hypoestrinism—Secondary to Pituitary Hypofunction—Case with Primary Amenorrhoea—No Evidence of Complete Pituitary Hypofunction.

The patient, aged twenty, complained of primary amenorrhoea. She was engaged to be married. Puberty appeared at the normal time and her breasts developed normally. She had periodic molimina consisting of a feeling of lower abdominal congestion. At no time has she ever had hot flashes.

On physical examination her breasts were somewhat underdeveloped; the mucous membranes of her vagina were definitely atrophic; the uterus was small. Hair distribution was normal. Her span was 4.6 cm. greater than her height. P A was negative; E was negative on two occasions.

Comment:—From the physical examination and the history, it was obvious that this patient had hypoestrinism but not complete absence of the hormone. The tests show that the hypoestrinism is secondary to a hypopituitary condition. Without the tests she was indistinguishable as regards diagnosis from patient 4. She differs from the pituitary dwarf in that only one function of the anterior pituitary is involved (compare also patients 7 and 8 in this respect).

Patient 6. Hypoestrinism—Secondary to Pituitary Hypofunction—Associated with Gain in Weight.

This patient, aged twenty-seven, consulted the Clinic in July, 1933, because of amenorrhoea of five

and one-half years' duration. She started to gain weight at about the time of her marriage eight years previously, increasing from 130 pounds to 190 pounds three years before admission, since which time she lost thirty pounds by diet. A child was born shortly after marriage. About one year later she began to miss periods and they finally stopped altogether. She had had very few if any hot flashes. Pelvic examination showed atrophied mucous membranes as seen after the menopause and a small uterus. It was impossible to obtain an endometrial biopsy confirming the fact that the endometrium was atrophied. Roentgen examination showed a normal pituitary fossa. The basal metabolic rate was plus five.

The tests were as follows:

Date	E	P A
7-17-33	0	Neg.
10-23-33	0	
11-1-33		Neg.
12-4-33	0	

The negative E test on December 4, 1933, was immediately after ten daily injections of 100 units of antophysin, making it appear unlikely that such treatment would be of help in this case (v. discussion under "Treatment").

Comment:—Without the tests, one might have considered the patient as suffering from menopause praecox (cf. patient 3). The lack, or almost lack, of hot flashes was of course against such a diagnosis. This patient's pituitary lack seemed to be confined to the gonad stimulating hormone. Whether obesity is cause or effect is hard to say, but the former is more likely in spite of the fact that reduction of weight was without benefit in this case.

Patient 7. Hypoestrinism—Secondary to Pituitary Hypofunction—Sudden Unexplained Onset.

The patient was first seen at twenty-five. Her catamenia started at twelve and one-half and was perfectly normal until twenty. She then had a scanty period following which she never had any more periods. She had no molimina of any kind following cessation of periods and no hot flashes. There was no important change of diet which could be elicited to account for sudden cessation. The history is unquestionably correct and complete.

On physical examination she was an exceptionally well-developed and healthy-appearing individual with a normal feminine figure. Hair distribution was normal. Breasts were well developed and firm. Pelvic examination showed a very small clitoris, a very small cervix and a uterus very small and freely movable. The smallness of the uterus was verified by lipiodol injection. The mucous membranes of the vagina were pinkish rather than slightly purplish.

The hormone tests repeatedly showed E = O; P A = O. E remained negative in spite of a thorough trial of treatment with the gonad stimulating hormone of the urine of pregnant women.

Comment:—An unusual case apparently due to a sudden loss of prolan A production in the pituitary in a person who previous to sudden onset was perfectly normal. Other pituitary hormones were not involved.

Patient 8. Secondary Amenorrhoea. Simmond's Cachexia.

This patient, a married woman of 28, was first seen in October, 1933. She complained of amenorrhoea, sterility, headaches, loss of strength and loss of weight. Six years previously her periods became irregular and three years previously a cystic left ovary was removed because of left lower quadrant pains. She had no more periods except for one three months previously.

On physical examination her blood pressure was persistently low (90/70); the mucous membrane of the vagina was atrophied; cervix and uterus were small.

X-ray showed an enlarged sella. The basal metabolic rate was minus 21. Urine tests: P A = Neg. E = O (repeatedly).

Comment:—This patient's amenorrhoea is clearly of the hypopituitary type. In contrast to the findings in some of the other patients, in this patient all the hormones of the anterior pituitary are apparently deficient (cf. low metabolism as evidence of decrease in thyreotrophic hormone, low blood pressure and asthenia, as evidence of decrease of adrenotropic factor). This patient's metabolism was elevated to plus four temporarily with the thyrotropic hormone and roentgen treatment of the pituitary resulted in marked clinical improvement.

Patient 9. Hypoestrinism—Primary Pituitary Hypofunction—Onset after Pregnancy.

This patient, aged twenty-seven, complained of sterility. Her catamenia began at twelve and was regular and normal in every way until after the birth of her only child in 1929. She nursed this child for six weeks, following which she had one period, but has had none since that time. She has had, to be sure, periodic molimina from time to time. Libido has remained normal.

Physical examination was entirely normal. The uterus was normal in size. The external genitalia were normal. There was no evidence of hypothyroidism.

Hormone tests: E = O (three times); P A = O (two times).

Comment:—The fact that this patient had molimina and normal-sized uterus made it obvious that her ovaries were not entirely without function; merely on the low side. The tests made it quite clear that she is primarily hypopituitary rather than hypo-ovarian. Pregnancy was probably an etological factor.

Patient 10. Hypoestrinism—Secondary to Pituitary Hypofunction—Onset Following Inadequate Diet.

This patient consulted the Clinic in May, 1933 because of amenorrhoea. Her periods were regular until May, 1931 when she considered herself too fat and went on a "strict diet". Her flow was scanty during the next two months and then ceased altogether except for scanty periods in July and October, 1932, following theelol injections. She has had some suggestion of hot flashes. In spite of the fact that she has given up dieting, periods have not returned.

On physical examination she was well developed; anthropometric measurements were normal. Hair distribution was normal. Skin was dry. Uterus was normal in size. Basal metabolic rate was minus twenty-six and minus twenty-seven. Weight

was ninety pounds, eighteen pounds below her best weight.

Hormone tests: P A = Neg.; E = O.

Comment:—This is a very frequent type of case and presumably attributable to a damage in prolan A production by the pituitary due to insufficient building blocks. In some cases this damage is permanent; more often not. The low metabolism is likewise frequent. It may return to normal with adequate diet although one of our patients similar to this had to take thyroid indefinitely.

III. PERSISTENT ESTRIN FORMATION—WITHOUT PROGESTIN FORMATION

There is no absolute proof that this group exists but it probably does. It would be analogous, somewhat, to what occurs in rabbits. These animals when in heat can remain for months at a time with the ovarian follicles mature and the endometrium built up to its full estrin development. They require the added stimulus of copulation for ovulation and the development of corpora lutea and the progestin phase. In the condition in human beings called metropathia hemorrhagica, something quite similar probably occurs. Long periods of amenorrhoea followed by uterine bleeding characterize this condition. The bleeding is probably not true menstruation but sloughing of the hypertrophied endometrium so that even when bleeding these patients in a sense are amenorrhoeic. Their ovaries show evidence of estrin formation, but no evidence of progestin formation. The changes in the endometrium are consistent with a long-standing estrin effect. Their hormone tests usually show P A = Neg., E = +. There is no indication from the E tests that this condition is a hyperestrinism. The evidence favors, we believe, an uninterrupted estrin effect. This condition, however, is so important that its handling is being reserved for a separate paper and for the collection of further data.

The following two case histories are of interest as possible examples of persistent estrin formation of a slightly different variety.

Patient 11. Persistent Estrin Formation—Infrequent Periods—Change to "Rabbit-form of Cycle".

This patient, a married woman of thirty, complained of sterility and infrequent catamenia. She was first seen on June 28, 1934. Periods began at thirteen; had always been infrequent; lasted about seven days. Between periods she had a constant white vaginal discharge. Her last period began on May 4; the previous one November 16, 1933. She had been married three years but had never become pregnant, in spite of the fact that for the past year she has been very anxious to have children.

On physical examination she appeared exceptionally well. She had a very normal female figure. The breasts were very well developed. The mucous membrane of her vagina showed no evidence of atrophy; the uterus was good sized.

The impression at this point was that this patient's amenorrhoea was not hypo-hormonal. From the examination of her uterus and vagina it seemed certain that she was producing a normal amount of estrin, as long-standing lack of estrin causes regression of the myometrium and atrophy of the vagina. The tentative hypothesis was formulated that she ovulated only infrequently but between periods was, during the majority of the time, in a constant state ready to ovulate. This is about what takes place in rabbits which remain at the height of estrus with ripened follicles until the added stimulus of copulation causes ovulation. Such a hypothesis would explain this patient's continued white discharge, as estrin in large amounts causes such a discharge.

One had to make the further assumption that coitus in this patient, unlike the situation in rabbits, was not followed by ovulation. This led to the suggestion that an endometrial biopsy, a procedure which probably is often followed by ovulation, might cause ovulation and that coitus after this procedure might result in pregnancy. The suggestion that an endometrial biopsy is apt to be followed by ovulation was made to us by Burch[10].

On July 5, P A was negative; E showed twenty units, confirming the suggestion that this patient was producing estrin. An endometrial biopsy done on this same day showed definite evidence of estrin effect—mitotic figures in the glands, but no secretory activity. Within several weeks the patient developed symptoms of pregnancy and such a diagnosis was confirmed by an Aschheim-Zondek test. Thus all the data seem to fit the hypothesis suggested.

Patient 12. Persistent Estrin Formation—Amenorrhoea—A "Rabbit-Form of Cycle"—Partial Sterility.

This patient of twenty-nine, married three years, consulted the clinic September 26, 1932. She had had periods every four weeks from the ages of 15 to 19 but for the past eight years only every six to eighteen months. They were rather profuse and lasted eight days. She had no periodic molimina. Examination showed a rather obese woman with well-developed breasts and a feminine configuration. She had a slight amount of hair on the upper lip and abundant, though normal amount of pubic and axillary hair. The clitoris was moderately hypertrophied and the uterus and cervix were normal. One observer thought the right ovary was enlarged.

E was 14 units and 35 units on April 8 and 9, 1934 respectively. P A was negative on April 14, 1934.

She had but one period from September 1931 to January 1934, but during this interval she became pregnant without any specific treatment.

An endometrial biopsy, taken after four months of amenorrhoea, showed a marked estrin effect but no secretory activity at all. Ten days after the biopsy she had a normal period.

Comment:—The facts in this patient suggest a similar interpretation as that in patient 11. We believe that during the amenorrhoea phase she has a ripe follicle putting out estrin (cf. biopsy and E tests). The biopsy, we believe, led to ovulation, followed by a corpus luteum which stopped functioning at the normal time, with resulting menstruation. The fact that she became pregnant shows that eventually spontaneous ovulation occurs.

IV. PERSISTENT ESTRIN AND PROGESTIN FORMATION

The amenorrhoea of pregnancy and perhaps that of lactation are the obvious examples in

this group. There are in addition a few reports in the literature[11, 12] where a woman misses one or more periods; develops the symptoms and some of the signs of pregnancy; is examined and found to have a mass in one vault; is operated upon for a presumable ectopic pregnancy; is found to have a corpus luteum cyst instead of an ectopic pregnancy; and lactates following the removal of the cyst. On one such patient a false positive Aschheim-Zondek test for pregnancy was obtained[13]. A very interesting patient reported by Wagner later developed definite findings of a pituitary tumor[14].

We have not had an opportunity to study any such patient but the tests should be the same as during pregnancy, namely P A = ++; E = +++. In addition there would be a positive prolan B (P B) test.

V. ENDOMETRIAL ABNORMALITIES

Obviously if there is a developmental absence of the uterus one might have normal hormonal factors and still amenorrhoea. The same would be true following hysterectomy. Patient 13 is probably an example of maldevelopment of the uterus.

Patient 13. Primary Amenorrhoea—Normal Hormone Tests—Fault with Uterus.

This patient, aged nineteen, never had a period. The breasts were well developed. It was impossible to do a vaginal examination, but by rectum the uterus could not be felt. Hair distribution was normal. The ovaries were not palpable. The tests showed:

Date	E	P A
6-7-33	28 units	
7-10-33		0
7-30-34	26 units	0

Comment:—The tests confirmed the clinical impression that this patient was normal in respect to the hormones and that the trouble was entirely developmental.

BORDERLINE CASES

A discussion of certain cases bordering on the normal but probably belonging in groups I and II has been postponed until here. In group I the possibility exists for what might be termed a partly compensated hypoestrinism. Thus, a hypofunctioning ovary might be stimulated by an excess of Prolan A to produce detectable amounts of estrin. This might lead to the findings:—E = +, P A = + (see patient 14). Presumably more accurate measuring sticks will show that E is really diminished in such cases. In a similar manner there are undoubtedly patients in group II who only have a partial hypofunction and whose tests might occasionally show E to be positive (see patient 14). A single pair of determinations might, therefore, be consistent with normal. Repeated E tests, however, would probably show estrin excretion to be decreased.

Patient 14. Hypoestrinism—Primary Ovarian—Partially "Compensated".

This patient, a trained nurse of twenty-two, complained of amenorrhoea. She had had only one catamenia (October, 1933) during her entire life. She had had no periodic molimina. Puberty occurred at fifteen.

On physical examination she had an only incompletely developed feminine figure; her breasts were only moderately well developed. Hair distribution was normal. The cervix and uterus were small. Her anthropometric measurements showed a disproportionate length of her long bones. Her basal metabolic was minus six. Her tests were as follows:—

Date	P A	E
June, 1934	Slightly Positive	0
July, 1934	" "	38 units
Sept., 1934	Negative	0

Comment:—From the physical examination and the history it was evident that this patient had hypoestrinism but not complete lack of this hormone. The finding of two negative E tests out of three was consistent with this. The P A tests likewise suggest only a moderate overactivity of the anterior pituitary.

Patient 15. Hypoestrinism—Secondary to Pituitary Hypofunction—Obesity.

This patient consulted the Clinic in July, 1934, at the age of sixteen because of amenorrhoea. Her catamenia began at thirteen, but has never been regular. She has always been overweight. At one time with reduction of weight by diet her periods became quite regular, only to cease again after discontinuing the diet and gaining more weight. At the time of admission she had had no periods for six months.

On physical examination she was very obese; palms were warm and moist and there was no suggestion of hypothyroidism; breasts were well developed; external genitalia were normal.

The admission impression was that she had hypoestrinism, secondary to hypopituitarism, secondary to obesity. It was also clear that she was not entirely lacking in estrin. Tests:—

Date	E	P A
8-6-34	10 units	Negative
9-7-34	0	Negative

Comment:—The findings fit in with the diagnosis of hypoestrinism, on a hypopituitary basis in spite of the fact that one E test was positive.

TREATMENT

The subject of treatment is too long and as yet too uncertain to be discussed here in full. A few points seem pertinent, however. In the first place it is important to keep in mind just what one is striving to obtain. There is no use, except for psychological reasons, in bringing back "the curse", for example, if the patient's only complaint is sterility and if the measures employed to bring back the catamenia could in no way influence the sterility. One can be certain, however, that intelligent treatment in the future is going to rest on an accurate diagnosis of just what link is at fault. For example, administration of gonad stimulating substances is illogical when the patient's own

pituitary is already producing an excess of prolan A to which the ovary will not respond. Furthermore, an ovary which is being understimulated and is putting out neither ova nor estrin will not be stimulated to so doing by estrin treatment. Such therapy is like expecting thyroxin to stimulate the thyroid to put out the thyroid hormone. The measuring sticks will, therefore, become very important, we believe, in deciding which specific treatment, if any, to institute. They will also probably be of help in estimating the success of treatment. Thus, if a gonad stimulating substance is being used one would be encouraged to its further use by the finding that estrin appeared in the urine and vice versa if it did not (cf. patients 7 and 8).

SUMMARY

On the basis of the hormonal measuring sticks the various types of amenorrhoea have been divided into three groups—hypo-hormonal, continuous hormonal and normal-hormonal. The first group has been further divided into a subgroup where the ovary is intrinsically at fault and one where the ovarian hypofunction is secondary to that of the pituitary. The second group is divided into a sub-group where there is constant estrin production and one where there is constant production of both estrin and progestin. Clinical examples of the five groups are cited.

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A COMPARISON OF "YEAST MILK" AND IRRADIATED MILK IN THE TREATMENT OF INFANTILE RICKETS*

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THE uncertainty existing in the minds of pediatricians and others regarding the relative clinical efficacy of vitamin D milks has been engendered by numerous factors. The misinterpretation often placed on a study by Hess[1] that irradiated milk of a given unitage has an antirachitic potency substantially as effective as twice that unitage in milk produced by feeding cows irradiated yeast, and the fact that commercial practice has restricted irradiated milk to a potency no higher than 55 units per quart, while yeast-fed cows' milk generally is marketed with a potency of 160 units per quart, have contributed to the confusion. A comparative study of the two types of milk, each with the same rat unitage, and administered in substantially equal amounts, should add information helpful in solving this question. At the time of the inception of the work herein reported, only one such study, that by Kramer and Gittleman[2], had been published. Their findings did not bear out the implications of Hess's results, referred to above. If further clinical tests should confirm the presumption that irradiated milk is clinically twice as potent, on the basis of rat units, as yeast-fed cows' milk, there would then be an adequate justification for the existing disparity in levels of potency of these two kinds of vitamin D milk. If, on the other hand, it should be shown that a given unitage of either kind of milk is equally efficient in human antirachitic therapy, it then seems probable that yeast milk with its present greater potency per quart offers more protection per unit of volume than does irradiated milk.

A review of previous clinical tests of vitamin D milk is omitted from the present report, since these have recently been analyzed and reported elsewhere[3].

The investigation reported at this time was designed to compare infants with rickets in the Infants' Hospital in Boston, under continuous supervision, by determining the actual measurable effects of prescribing to equal numbers of cases of definite rickets in infants, yeast milk or irradiated milk of equal rat unit potency in the amounts normally called for in feeding each case.

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The yeast milk was from a special herd*, fed a ration containing irradiated yeast in amounts calculated by the Fleischmann Yeast Company to produce a vitamin D milk of 50 rat units per quart. The irradiated milk* was to be of the same potency.

Since in a comparative study of this sort, it is necessary that the unitage of each type of milk be definitely known at all times, it is essential that frequent or reliable bio-assays of the milks be made throughout the duration of the test. In the present case, five bio-assays of each kind of milk, with numerous International Standard parallel evaluations, were carried out at the Biological Laboratories of the Massachusetts Institute of Technology during the twelve weeks of the clinical investigation. Sealed quarts of the respective milks in each case were secured from the hospital immediately before assay. Each specimen was fed in suitable dilutions of whole milk to a group of ten albino rats, previously rendered rachitic, with due regard for weights of animals and food consumption, during the first eight days of a ten-day test period. On the tenth day, the degree of healing of rickets was determined by x-ray and line test. An equal number of control animals was held without antirachitic supplement during the entire period and subjected to similar diagnoses. The results of these findings are presented in table 1.

TABLE 1

RESULTS OF BIO-ASSAYS OF YEAST MILK AND IRRADIATED MILK TOGETHER WITH INTERNATIONAL STANDARD EVALUATIONS

Date of Sample	Steenbock Rat Units per Quart*		Equivalence of One† Steenbock Unit in International Units
	Yeast Milk	Irradiated Milk	
Feb. 12	More than 85	Barely 50	
Mar. 1	More than 60	Less than 50	
Mar. 19		Less than 50	
Mar. 20			2.7
Mar. 25	65		
Apr. 3			2.7
Apr. 19	60	55	
Apr. 21			2.7
May 10	65+	Barely 50	
May 11			2.7

*Wistar albino rats, 28 days old, 50-55 grams, were rendered rachitic on Steenbock No. 2965 ration, containing specially selected and aged corn[4]. Whole milk, suitably diluted with water and pipetted into separate jars, was fed in 8 daily portions of 5 cc. per rat.

†International Standard Reference Oil, diluted gravimetrically with olive oil, was dispensed in calibrated drops directly into the mouth of each animal during eight days.

Since the initial assay of yeast milk showed too high a potency for this comparison study, the yeast ration of the cows was adjusted and the infant feeding of this milk deferred until subsequent assay showed a potency more nearly that of the irradiated product. While not pertinent to the present discussion, it may be of passing interest to note that at the lowered levels of yeast feeding of cows, a mathematical reduction of the yeast ration was not followed by an arithmetically proportionate reduction in the antirachitic potency of the milk produced.

*H. P. Hood & Sons, Inc., Boston.

OUTLINE OF CLINICAL EXPERIMENTS

A brief history of the six infants with active rickets who were selected for this study is presented, together with significant findings. On admission to the hospital the patients were placed in a separate ward and during a preliminary period each was given a simple calorifically adequate diet, without antirachitic supplement, to make certain that healing was not taking place. This was determined by roentgenograms of the right wrist and by determinations of the calcium and inorganic phosphorus content of the blood serum at regular intervals. The sera were separated immediately after being taken, and the inorganic phosphorus determined[5] at once. Two cc. of serum were used for each calcium determination[6]. Having satisfied ourselves by these preliminary observations that healing was not taking place, the milk in the diet of three infants was replaced by irradiated milk, and that of the other three infants was replaced by yeast milk. During the test period roentgenograms and blood analyses were frequently made so that the progress of the treatment might be closely followed.

CLINICAL RESULTS

Irradiated Milk

CASE 1.—R. C., an Italian boy, 6 months old, weighing 15 pounds 8 ounces, was admitted to hospital January 13, with the diagnosis of active rickets and eczema. He had been breast-fed for 1½ months, and since then had been fed whole milk, water and maltose mixture. He had never received antirachitic treatment and had been given orange juice infrequently. Physical examination showed a fairly well-developed and nourished infant who was very uncomfortable and irritable because of eczematous rash on the face. The remainder of his skin was clear except for roughness of hands. Craniotabes, Harrison's groove, rachitic rosary and enlarged epiphyseal ends of long bones were present. On admission he was placed on a diet of evaporated milk, Karo corn syrup and water, cereals, vegetables and orange juice. Behavior during the preliminary period and the history of the case indicated that

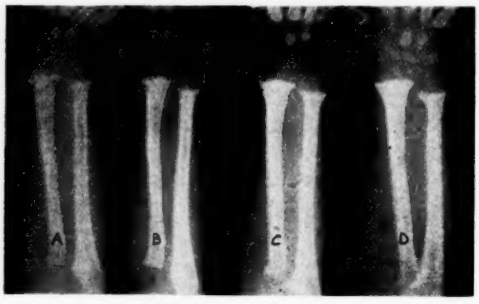

FIG. 1 (Case 1) R. C.: A = January 15, advanced rickets; B = January 24, no change; C = February 15, healing after 22 days on irradiated milk; D = March 10, further healing after 45 days.

the patient had active advanced rickets. Irradiated milk was substituted for the evaporated milk on January 24. The data for this case are summarized in table 2.

CASE 2.—N. L., a colored boy, 7 months old, weighing 19 pounds 14 ounces, was hospitalized February 13 because of rash on the face (eczema) and active rickets. He had been wholly breast-fed and had grown rapidly. Orange juice had been given occasionally and ½ teaspoon of cod liver oil had been given irregularly during the past month. Physical examination showed a well-developed and nourished Negro, with a dry scaling eczematous rash over the forehead, back, arms and legs. Prominent bosses, a rachitic rosary, Harrison's groove, and enlarged epiphyseal ends of the long bones were present. On admission he was given the ward diet mentioned in the history of Case 1. During the control period the CaxP product rose from 29.4 to 39.1 but the roentgenograms showed no detectable change. The case was considered suitable for the study, but not entirely satisfactory. Irradiated milk was substituted on February 24. The data are summarized in table 2.

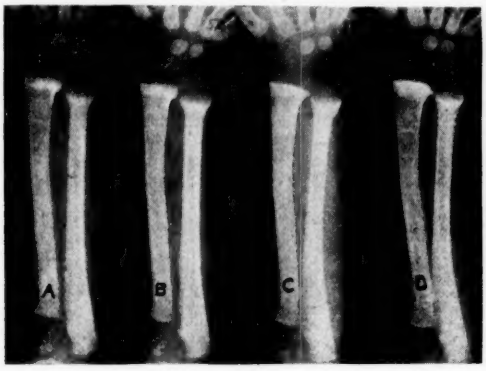

FIG. 2 (Case 2) N. L.: A = February 12, advanced active rickets; B = February 19, no change; C = March 2, slight healing after 6 days on irradiated milk; D = March 23, further healing after 27 days.

CASE 3.—C. F., a colored boy, 26 months old, weighing 21 pounds 3 ounces, was hospitalized April 2 because of inability to walk, with the diagnosis of active rickets. He had been breast-fed during the first three months, had been given condensed milk during the next four months and then had been given general diet. During the first 18 months he had received two teaspoons of cod liver oil daily and had never had orange or tomato juice. Since then he had received no antirachitic supplement. At seven months he sat up but to date had not walked. During the last three months he had cried with pain when handled. Physical examination showed a well-nourished colored boy, small for his age, with prominent frontal bosses, rachitic rosary, flaring thorax, Harrison's groove and enlargement of the epiphyseal ends of the long bones. After two weeks on ward diet, roentgenograms showed no change, although there had been a rise in serum phosphorus. Irradiated milk was substituted on April 18. On April 30 he developed measles from which there was an uneventful recovery. During this illness he was removed to the isolation ward but the same feeding régime was continued. The data for this case are summarized in table 2.

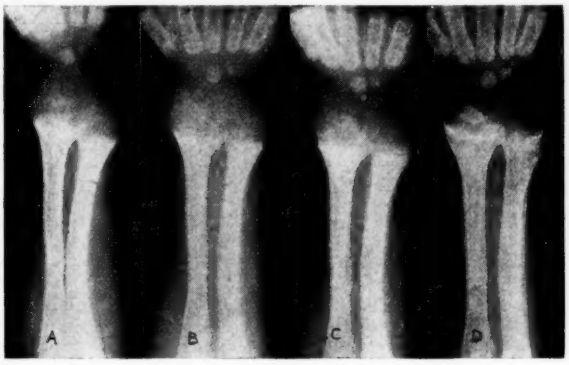

FIG. 3 (Case 3) C. F.: A = April 2, advanced chronic rickets; B = April 16, no change; C = May 10, healing rickets after 22 days on irradiated milk; D = May 29, further healing after 41 days.

TABLE 2

SUMMARY OF CLINICAL CASES

(*Irradiated Milk*)

Case	Date 1934	Weight lbs.-oz.	Milk oz./day	Calories per lb.	Serum (mg. %) Ca	Serum (mg. %) P	CaxP Product	Roentgenograms
No. 1	1/15	15:8	23	48.6	7.8	3.9	30.42	Advanced active rickets
R. C.	1/23	15:8	23	48.3	6.3	3.5	22.05	No change
Age	1/24				*Irradiated milk started*			
6 mos.	2/1	15:7	26	49.6	11.7	3.4	39.78	No change
plus	2/10	16:4	28	52.2	11.2	4.4	49.28	No change
	2/16	16:11	28	51.2	9.9	5.7	56.43	Slight healing
	2/24	17:3	28	49.3	11.1	6.0	66.60	Further healing
	3/5	17:15	28	47.0	11.8	6.8	80.24	Further healing
	3/10	17:12	28		10.7	6.5	69.55	Further healing
No. 2	2/14	19:14			9.2	3.3	30.36	Advanced active rickets
N. L.	2/21	19:2	20	36.7	10.3	3.8	39.14	No change
Age	2/24	19:3	20	37.0	*Irradiated milk started*			No change
7 mos.	3/2	18:14	20	38.5	9.8	4.0	39.20	Slight healing
	3/10	18:10	20	34.0	9.9	3.5	34.65	Very little change
	3/17	19:3	26	38.8	8.6	4.7	40.42	Healing
	3/23	19:7	26	38.2	11.2	5.8	64.96	Further healing
No. 3	4/2	21:3	32		10.1	2.3	23.23	Advanced chronic rickets
C. F.	4/9	21:8	32		10.7	3.3	35.31	No change
Age	4/16	21:13	32		10.5	3.5	36.75	No change
26 mos.	4/18				*Irradiated milk started*			
	4/23	21:14	32		10.4	3.5	36.40	No change
	5/7		32		9.7	3.7	35.89	Healing rickets
	5/22		32		10.5	5.3	55.65	Further healing
	5/28	22:12	32		10.3	5.7	58.71	Further healing

Yeast Milk

CASE 4.—J. S., a Greek boy, 8 months old, weighing 10 pounds 14 ounces, was admitted on March 5 with a diagnosis of rickets. A premature baby with a birth weight of 4 pounds and 11 ounces, he had been cared for in a premature nursery for two months. Since then he had been on a well-balanced

FIG. 4 (Case 4) J. S.: A = March 6, advanced active rickets; B = March 19, no change; C = March 31, healing rickets after 12 days on yeast milk; D = April 28, further healing after 40 days.

diet of cow's milk and cereal supplemented with one ounce of orange juice daily. Four months before admission his mother was told that he had rickets and was advised to give him cod liver oil. This had been offered a few times but was discontinued because he refused his food. Physical examination showed a poorly developed and nourished, pale infant with prominent frontal and parietal bosses, craniotabes, rachitic rosary and enlarged epiphyseal ends of the long bones. On admission he had an acute upper respiratory tract infection. Because of the definite absence of antirachitic therapy in the history and because of the absence of signs of rachitic healing during the preliminary period on the ward diet, this patient was considered suitable for the study. Yeast milk was substituted on March 19. The data for this case are summarized in table 3.

CASE 5.—L. Z., an Italian girl, 9 months old, weighing 11 pounds 5 ounces, was admitted March 15 with the diagnosis of active rickets. A premature baby with a birth weight of 3½ pounds, she had remained in premature nursery care for four months. Since then she had been on a well-balanced formula of cow's milk. No other food had been given. Cod liver oil had been given from the fourth to

FIG. 5 (Case 5) L. Z.: A = March 15, advanced active rickets; B = March 26, no change; C = April 10, slight healing after 22 days on yeast milk; D = April 16, further healing after 28 days.

the eighth month, 10 to 12 drops per day. Physical examination showed a small, pale infant with a profuse mucopurulent discharge, craniotabes, marked flattening of the right occiput, Harrison's groove, rachitic rosary and enlarged ends of the long bones. Cultures from the nose and throat showed virulent Klebs-Loeffler organisms and she was given 10,000 units of diphtheria antitoxin. Previous history and behavior during the preliminary period on the ward diet indicated that the case was suitable for the study. Yeast milk was substituted on March 19 and the study was continued in the isolation ward. The data for this case are summarized in table 3.

CASE 6.—P. R., an American girl, 22 months old, weighing 18 pounds 2 ounces, was admitted on April 3 with a diagnosis of active rickets with bony deformities. She had been breast-fed for 12 months and had since been on a baby soft diet. She had received orange juice occasionally and had never been given antirachitic supplements. At fourteen months her mother had noticed that she did not

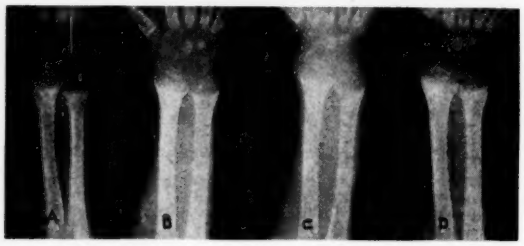

FIG. 6 (Case 6) P. R.: A = April 3, advanced chronic rickets; B = April 9, no change; C = May 7, slight healing after 27 days on yeast milk; D = May 29, further healing after 49 days.

creep or try to walk as had her other children. Physical examination showed a well-nourished infant with prominent frontal bones, flaring thorax, rachitic rosary, Harrison's groove and enlargement of the epiphyseal ends of the long bones. During the preliminary period on the ward diet the case showed no healing. On April 10 yeast milk was substituted. Data for this case are summarized in table 3.

DISCUSSION

Six hospitalized cases of definite active rickets showed healing within four weeks when fed 26 to 32 ounces of vitamin D milk daily, irrespective of whether it was irradiated milk of 50 units or yeast-fed cows' milk of 60 to 65 units per quart. The cases included two Negroes, two Italians, one Greek and one American, and two of the group were premature infants. From examination of the serum CaxP products, and from the roentgenograms, there is no obvious difference in the clinical antirachitic value of the two types of milk, as shown by the cases reported.

In attempting to compare further the relative effects of human infant consumption of the two types of vitamin D milk employed in this study, it seemed useful to plot against time for each case studied, the product of the serum Ca and serum P. The result is displayed in fig. 7. All cases showed a CaxP product of less than 40 when the vitamin milk was first administered. The progressive increase of this product throughout the duration of the test is indicated. The general trend of the curves is substantially the same for each group. If the rate of increase of the serum CaxP product is an index of the rate of recovery from rickets, there is

nothing to choose between the rates of recovery on the two kinds of milk used.

A comparison of similarly plotted values of

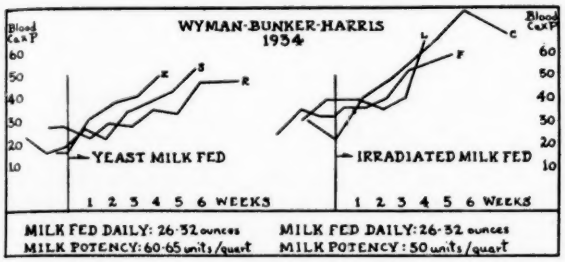

FIG. 7.

serum CaxP from the Kramer and Gittleman[2] study of infants on the two kinds of vitamin D milk, of approximately the same potency as used by us, supports the same conclusion, the trend of the curves (fig. 8) being substantially

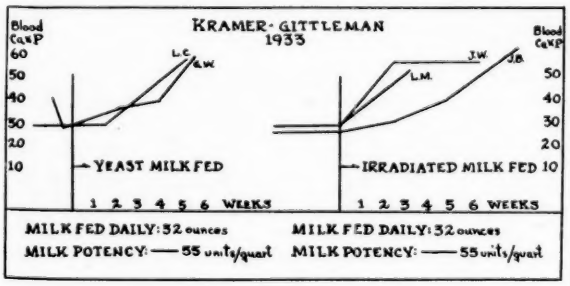

FIG. 8.

the same in both of their groups and in each of ours.

It is the opinion of one of us (E. T. W.) that the initial response of the rachitic infants to the administration of vitamin D milk in the present study is slower than that of similar infants in a previous study[9] where yeast milk of a higher potency was given. If serum CaxP values are truly an index of the healing process in recovery from rickets, then the above observation should be supported by the plot of these values for the earlier test (fig. 9) when compared with a similar plot for the present study (fig. 7). Such comparison fails to indicate to us the expected difference in the general slope of the curves. However, it is realized that since the number of cases is much too small to permit of calculating the probable error, it is impossible to give proper weight to a single curve which departs from the apparent mean (cf. case D. E., Fig. 9, and case J. B., Fig. 8). It is quite possible that a sufficiently large number of records of serum Ca and P determinations would permit a more definite evaluation of such findings.

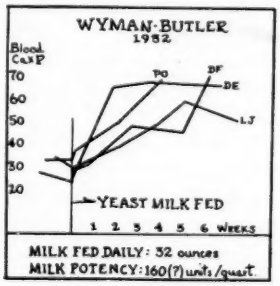

FIG. 9.

TABLE 3

SUMMARY OF CLINICAL CASES

(*Yeast Milk*)

Case	Date 1934	Weight lbs.-oz.	Milk oz./day	Calories per lb.	Serum (mg. %) Ca	Serum (mg. %) P	CaxP Product	Roentgenograms
No. 4	3/5	10:14	28	64.0	11.0	2.1	23.10	Advanced active rickets
J. S.	3/12	10:13	26	70.4	9.0	1.8	16.20	No change
Age	3/17	11:1	26	68.3	7.5	2.6	19.50	No change
8 mos.	3/19	11:2	26	68.0	*Yeast milk started*			No change
	3/23	10:13	26	67.7	9.8	2.8	27.44	No change
	3/30	10:10	26	68.0	9.3	2.4	22.32	Healing rickets
	4/6	10:12	26	67.2	9.9	3.4	33.66	Further healing
	4/13	10:14	26	66.6	9.5	4.0	38.00	Further healing
	4/21	10:12	26	67.7	10.2	4.2	42.84	Further healing
	4/28	10:13	26	67.7		5.3		Further healing
No. 5	3/15	11:5	17	47.9	8.2	1.9	15.58	Advanced active rickets
L. Z.	3/19	11:14	17	48.2	*Yeast milk started*			No change
Age	3/26	12:2	26	53.0	10.1	3.0	30.30	No change
9 mos.	4/4	12:8	26	51.0	11.8	3.2	37.76	Slight healing
	4/10	12:14	28	54.0	11.1	3.6	39.96	Further healing
	4/16	13:10	28	51.0	10.5	4.7	49.35	Further healing
No. 6	4/4	18:2	32	42.5	10.9	2.5	27.25	Advanced chronic rickets
P. R.	4/9	18:2	32	42.5	11.0	2.5	27.50	No change
Age	4/10	18:2	32		*Yeast milk started*			No change
22 mos.	4/16	18:3	32	44.2	9.8	2.2	21.56	No change
	4/23				10.2	2.9	29.58	No change
	4/30	18:12	28	42.6	9.9	2.8	27.72	No change
	5/7	18:9	28	43.0	10.4	3.4	35.36	Slight healing
	5/14	18:15	28	42.5	10.4	3.2	33.28	Further healing
	5/22	18:15	28	42.5	11.0	4.3	47.30	Further healing
	5/28	19:2	28	41.8	10.1	4.7	47.47	Further healing

It seems fair to conclude, however, that with a small number of cases, and especially in a single case, the product of serum calcium and serum phosphorus, as ordinarily determined, cannot be regarded as a reliable quantitative expression of either speed or degree of healing. For the present, such evidence should be regarded as merely contributory to a complete diagnosis.

It may not be amiss in this connection to refer to the experimental work of Robison and Soames[8] on the calcification of split bones from rachitic rats in solutions containing salts of calcium and phosphorus. These authors found that when the menstruum contained Ca and P ions in proper proportions to give a product of about 40 (the calcium being in concentration of about 10 mg. per cent), some calcification from inorganic salts could be obtained in the rachitic metaphyses, but that traces of organic phosphoric esters greatly enhanced the calcification and even permitted it to take place at lower CaxP values. It is interesting that the CaxP product required for calcification in rachitic bones in these experiments in vitro should so closely approximate the value expected in the human serum if bone calcification is to take place[7]. This work suggests also the possibility that determination of organic rather than inorganic phosphorus, which the present method[8] gives, might constitute an improvement in this diagnostic procedure. The question can be settled only by the accumulation of sufficient data from parallel determinations of both forms of phosphorus in the serum of rachitic and nonrachitic infants.

SUMMARY

Six infants with active rickets were fed 26 to 32 ounces of either irradiated milk (50 Steenbock Units per quart) or yeast-fed cows' milk (60 to 65 Steenbock Units per quart) during the early spring of 1934. All showed rachitic healing.

There was no indication of any difference between the two milks, unit for unit, in respect to their clinical antirachitic effectiveness.

Dr. Allan M. Butler directed the calcium and phosphorus determinations for the study and Dr. E. C. Vogt coöperated in taking the many roentgenograms and gave advice in their interpretation.

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LESS LEAD PERMITTED ON APPLES AND PEARS

In a notice addressed to both consumers and producers of apples and pears, Secretary of Agriculture Wallace has announced a further restriction in the quantity of lead residue permitted on these fruits in interstate commerce. The tolerance specified for the crops of 1935 is 0.018 grain of lead to a pound of fruit. The reduction was made despite requests from some quarters that the restrictions be relaxed, the Secretary adding that "expectation of continued progress downward is fully justified" and "relaxation in the lead restriction would be wholly incompatible with public safety".

The Food and Drug Administration will proceed against violators of the restrictions. Other spray residues for which tolerances are enforced are those containing arsenic or fluorine. The fruit industry, Secretary Wallace says, has demonstrated its ability to reduce the arsenic content to the world tolerance of 0.01 grain of arsenic trioxide to a pound of fruit. The official notice continues this tolerance for 1935. The fluorine tolerance also remains at this same point (0.01 grain to a pound of fruit). The reduction on lead is from 0.019 to 0.018.

The Department of Agriculture is continuing its efforts to find a safe and effective substitute for poisonous spray materials, but lead arsenate is still considered essential to orchard management. The Food and Drugs Act imposes on the producer the obligation to reduce the residue to the lowest possible point.

In his official notice Secretary Wallace says, "Consumers are entitled to know that the restrictions imposed for many years against the shipment of fruit subjected to poisonous sprays have been so effective that unquestionably to-day most of the fruit offered the public is entirely safe." He adds further that since the effects of lead and arsenic are chronic rather than acute, the few instances in which interstate shipments of fruits with excess residue escape seizure by the Food and Drug Administration cannot be considered "significant health hazards".

Consumers who insist on being doubly sure are advised by the Department that it is unnecessary for them to use acid or alkali cleaning solutions such as apple and pear growers use to loosen residues for washing off. In the kitchen each individual fruit is usually washed anyway. Peeling or cutting out the stem and blossom ends, where the residue is most difficult to wash is an added measure of safety.—*U. S. Department of Agriculture.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21061

PRESENTATION OF CASE

A thirty-six year old American male office clerk entered complaining of weakness and edema.

Five months before entry he began to "lose his pep" and he consulted a physician who prescribed a tonic and a vacation. He spent two weeks in Maine and felt that he had gained strength. About four months before entry he noticed that his ankles had become swollen during the day and returned to normal when he awoke in the morning. He also noticed increasing shortness of breath, especially upon exertion, but no orthopnea. There was no pain or palpitation. A month later he began to have diarrhea—four or five loose, watery, occasionally clay-colored stools a day. There was no blood or mucus. A physician prescribed some pills which reduced the number of movements to two or three a day but the character remained unchanged. After about two weeks they became essentially normal. Two months before admission his ankles continued to be swollen even after a night's rest. His urine was again examined by his physician and this time a question of kidney disease was raised. He was put on a milk and cracker diet for three days and then the diarrhea returned. His urine was again examined and declared negative. Three weeks before admission he noticed fullness in his abdomen after eating only one-third of the usual amount. His diarrhea recurred but there was no nausea or vomiting. He stopped working for the first time in about fifteen years because of the marked edema of his legs, and weakness. Four days before admission jaundice developed without chills or fever. The jaundice gradually increased in amount.

At the age of four he had pneumonia with pleurisy following which he had an infection in his left hip which was incised and which drained for about one year. At the age of eight, twenty-eight years ago, he noticed a lump on a right rib which grew larger during the following year, reaching the size of a fist. Three ribs were removed with the tumor at a first class hospital and the diagnosis of fibrosarcoma was made. Since then many hard and soft lumps of varying size appeared on his head and other parts of his body. Several of these were removed and diagnosed as wens. His right chest gradually grew larger and became deformed. He had been able, however, to work as a clerk for about twenty years.

His father died in a sanatorium at the age of thirty-six of pulmonary tuberculosis. His mother died at the age of thirty-four from carcinoma of the rectum. One brother died at birth. A sister aged thirty-seven was living and well. The females in the family and their offspring for three generations back were afflicted with lumps similar to the patient's. His grandmother died of carcinoma.

His marital history is non-contributory.

Physical examination showed a very emaciated, markedly deformed man with a skeleton of unusual asymmetry. This was most marked in the thoracic region where an extreme right dorsal scoliosis was evident which made the right half of the chest appear approximately four times as large as the left. On further examination many other points of asymmetry became evident: the calvarium was markedly irregular with bony prominences over the left parietal and right occipital regions; hard, bony masses about 1.5 centimeters in diameter projected from each angle of the mandible, and the mandible itself seemed larger on the left than on the right. The nose, moreover, was distinctly more prominent on the left side and the septum was deviated to the left. The left hip was almost completely ankylosed and large irregular bony excrescences could be felt about the joint. Along the posterior aspects of the lower third of the right fibula two irregular projections of the bone could be felt, each about one centimeter in size. The thigh muscles of the left leg were distinctly atrophic in comparison with those on the right. Over this left hip was an old operative scar with a deep indentation suggesting a drainage wound. A second marginal incision was present on the posterolateral aspect of the right chest beneath which portions of three ribs were absent.

Scattered irregularly over the entire body, scalp, face, trunk and extremities were about 30 subcutaneous tumor masses varying from 3 millimeters to 3 centimeters in diameter. The smaller of these were firm, the larger frequently fluctuant. The skin was in some instances freely movable over the masses, in other instances was adherent to them. Over a few of the masses it appeared reddened, but over the majority it was unchanged in color.

The skin over the entire body seemed atrophic and slightly more transparent than normal. It was uniformly slightly but definitely icteric in tint and the sclerae showed definite jaundice. Over the lower right thorax and upper abdomen above the umbilicus numerous dilated cutaneous veins were apparent. A cluster of firm, discrete

cervical glands was felt below the left mandible.

The right pupil was greater than the left, but the reflexes were normal. Several punctate pigmented areas were noted in the fundi. The chest organs were markedly displaced by the scoliotic deformity but the heart seemed to be of normal size. Its sounds were of good quality, the rhythm was regular and the rate 110. The blood pressure was 112/70. A few fine crepitant râles were heard at the right base but the lungs were otherwise clear. On the right continuous dullness extended from the level of the fourth rib to a point 4 centimeters below the costal margin where the liver edge could be definitely felt. The abdomen was distended and tympanitic throughout. A long slender mass about 12 by 4 centimeters, feeling very much like a banana, extended horizontally across the abdomen at the level of the umbilicus. It was freely movable and non-tender. The scrotum was edematous. Rectal examination showed a ring of external hemorrhoids, and many others could be felt within the ring, which bled easily on palpation. There was gross pitting edema extending halfway up each leg.

The temperature was 102°, the pulse 120. The respirations were 26.

Examination of the urine showed a specific gravity of 1.020 to 1.022, a slight trace of albumin and a positive test for bile. The blood showed a red cell count of 3,330,000, with a hemoglobin of 55 per cent. There was a white cell count of 28,200, 90 per cent polymorphonuclears. A smear showed approximately 7 per cent myelocytes and 4 or 5 stippled red cells in about 15 high power fields. No parasites were seen. The platelets were normal. The stools were soft, clay-colored and although no blood could be seen grossly, all specimens showed a 4 plus guaiac test. A Hinton test was negative, the icteric index 50, the van den Bergh 13.87 milligrams per 100 cubic centimeters direct. The serum protein was 4.8 per cent. The liver function test showed 50 per cent retention. The serum calcium was 8.13 milligrams, the phosphorus 3.60, the cholesterol 155.

He ran a septic chart, his temperature rising to 103° and falling to 99 or 100°. His white blood cell count remained about 35,000 and his red cell count about 3,500,000. He gradually failed and died two weeks after admission.

Differential Diagnosis

Dr. Leland S. McKittrick: It certainly would seem that this story of a boy of eight, with a tumor of the ribs followed by multiple nodules, going on for a period of years, with an increasing deformity of his chest and then the development of weakness, edema, shortness of breath, then diarrhea, jaundice and temperature, finally death, should be a perfectly clear-cut clinical picture if we could properly put it together. It seems so sharply characterized that I cannot help feeling that it should be easy, but the more I think about it the more confused I become.

The past and family history are exceedingly interesting. The tumor of the right chest wall was diagnosed as fibrosarcoma twenty years ago. I believe I am right in presuming that the evaluation of cellular structures of tumors was not so highly refined then as now. Fibrosarcoma, as I know it, is largely a tumor of adult life and may be difficult to eradicate locally because of the marked tendency to recur; it metastasizes rarely, if at all, to regional nodes, occasionally to lungs or liver, and within a period of three to five years of operation, rather than after a longer interim. It would, therefore, seem unlikely that this so-called fibrosarcoma of twenty-eight years ago had anything to do with the symptoms for which he comes to the hospital at this time.

He apparently had two kinds of palpable tumors—(1) definitely in relation to the skin, some of which had been removed and diagnosed as wens, and probably were—(2) the other obviously in relation to the skeleton, involving the bones of the skull as well as the long bones. These have apparently been present since childhood. It is extremely interesting to note the presence of multiple tumors similar to those of the patient for three generations back. These tumors undoubtedly fall into a rather rare group of familial osteochondroma which, so far as I know, are benign, may be accompanied by marked deformity and might possibly be the etiological factor in the asymmetry which this patient presents; this would be additional evidence toward the contention that the present illness is a condition superimposed on the other and not the direct result of it. It is likewise difficult to connect the family history of tuberculosis with the patient's present illness.

It seems reasonable then to attempt to arrive at a more definite diagnosis by starting with his more recent symptoms and trying to evaluate a rather obscure, indefinite history superimposed upon a most unusual background. In brief, the story is one of weakness, edema of the ankles, shortness of breath, then diarrhea without blood, persistence and increase of the edema, a little fullness in the abdomen, then finally jaundice, the outstanding symptom apparently being the weakness and increasing edema. He comes to the hospital distinctly jaundiced, and has a banana-shaped, non-tender abdominal mass lying horizontally. He has a temperature of 102°, and a rather interesting smear, 90 per cent polynuclears, and 7 per cent myelocytes. Except for the abdominal mass, and an asymmetry of the pupils, and the deformity already discussed, he apparently has a definitely enlarged liver, a distended tympanitic abdomen, and marked edema of the scrotum and lower leg.

He also has some dilated superficial veins in the upper abdomen and lower right chest and some hemorrhoids which bled easily on palpation. He has a definite secondary anemia and a leukocytosis. He has a 4 plus guaiac in all of the stool specimens examined but no gross blood. Icteric index and van den Bergh are in keeping with his clinical jaundice. Serum calcium, phosphorus, and cholesterol give no helpful information. A serum protein of 4.8 per cent is low, below a level at which edema is expected to occur and undoubtedly a factor in the amount of the edema present. I should doubt very much that this alone would account for edema of such long standing and so marked below the level of the umbilicus and not elsewhere.

I cannot make a diagnosis from the above summary of positive findings. Hodgkin's disease at times, particularly in the later stages, may give fever, leukocytosis, a high polynuclear count, with an increased number of cells somewhat comparable to myelocytes. The only other evidence suggestive at all of Hodgkin's is the massive glands in his neck, and I should be quite willing to exclude this as a probable diagnosis. One would like to interpret the abdominal mass as a distended gall bladder. I have never seen a gall bladder occupy a transverse position in the abdomen, however. While the presence of progressive, painless jaundice, with a non-tender distended gall bladder would be strong evidence in favor of a malignant obstruction in relation to the ampulla, I am not able to interpret the mass as gall bladder much as I would like to.

While metastatic cancer of the liver may give a leukocytosis and a temperature up to 102°, there is nothing from the physical examination to suggest that the liver enlargement is due to metastatic disease. It would seem reasonable then to attempt to explain the edema and jaundice by a single process. This could then be explained by an infiltrating lesion obstructing the portal system, with resultant obstruction, not only of the common hepatic duct but of the portal vein and possibly the inferior vena cava. I should presume that this was most likely due to metastatic cancer, the origin of which I cannot say. I would, therefore, suggest as a diagnosis: cancer, primary source undetermined, with metastases involving the capsule of Glisson, possibly the retroperitoneal structures with pressure on the inferior vena cava.

Clinical Discussion

Dr. Tracy B. Mallory: This is obviously one of the most baffling cases we have had in the hospital for a number of years and the field is still wide open for suggestions.

Dr. McKittrick: It will not hurt my feelings for anyone to make a diagnosis, Dr. Mallory.

Dr. Mallory: Dr. Talbott, you saw this patient on the ward. Will you tell us what you thought about him?

Dr. John H. Talbott: The patient was a most unusual one as far as his history is concerned, and presented a most puzzling physical examination. There were two or three points that I was interested in. One was the hereditary factor as far as the nodules were concerned. This patient had had at least three immediate relatives who had these exostoses. We know that hereditary exostoses are present in the majority of the members of affected families, and males are affected three to one. In this instance it was the females, although we could not be certain of the history.

The scoliosis was another thing that interested me. I have never seen any more marked scoliosis than this patient had. It was a debatable point as to whether the removal of three ribs many years ago was responsible for producing a functional scoliosis, and I felt that at some time or other there was primary bone disturbance. When we come down to the serum calcium of 8.13 with a phosphorus of 3.6 I do not think that any emphasis can be placed on those figures. He had a serum protein of 4.8 and that will give you a calcium consistent with 8.1. On the ward there was little evidence of a disturbance of the phosphorus or calcium metabolism.

I have seen two cases of diffuse carcinomatosis in the absence of any obvious infection that had a persisting high white count, as this was, of 28,000 and 35,000.

Then the question arose as to whether these hereditary exostoses had become malignant. Ehrenfried has, I believe, the best series of cases and has gone over them very thoroughly. He made a survey of 500 cases in which about 5 per cent had become malignant. I think it is entirely possible that this patient had hereditary exostosis, and that at some time or other one had become malignant. He had an obvious malignancy in addition to the exostoses.

Dr. Richard H. Miller: Are you assuming that this family all had exostoses?

Dr. Talbott: I am. If he had exostoses in the head of the femur, where they frequently occur, and he had a history of tuberculosis, it is likely that he had a tuberculous hip that was aggravated by the exostosis that was already present. The fact that he did not have the typical bowing of the extremities that one sees in the congenital cases I do not believe is against the diagnosis that I have presented.

Dr. G. W. Holmes: Is the statement that Dr. Talbott has just quoted from Dr. Ehrenfried a generally accepted thing, that this type of exostoses may become malignant?

Dr. Mallory: I believe so, Dr. Holmes. It is uncommon, but it certainly does happen.

DR. MCKITTRICK: Was an x-ray taken of the chest?

DR. ALFRED KRANES: The x-ray of the chest showed a great encroachment on the right lung from the liver probably, and not much could be made out on account of the distortion from scoliosis.

DR. MCKITTRICK: There were no metastases?

DR. KRANES: No, sir.

DR. MAYO H. SOLEY: I examined the sister and she had almost the same situation in the jaw. We tried to get a report on a nephew of his, but were unable to do so.

CLINICAL DIAGNOSES

Carcinoma of the gastro-intestinal tract with metastases.
Hemorrhage into the gastro-intestinal tract.

DR. LELAND S. MCKITTRICK'S DIAGNOSIS

Carcinoma, primary source undetermined, with metastases involving the capsule of Glisson, possibly the retroperitoneal structures, with pressure on the inferior vena cava.

ANATOMIC DIAGNOSES

Multiple polyposis of duodenum, jejunum, ileum and large bowel.
Carcinoma at the papilla of Vater.
Acute cholangitis.
Abscess of gall bladder bed.
Icterus.
Multiple exostoses.
Multiple sebaceous cysts.
Operative wound, old: Resection of three ribs.
Functional scoliosis.
Pulmonary atelectasis.
Ascites.
Edema.
Secondary anemia.

PATHOLOGIC DISCUSSION

DR. MALLORY: The autopsy findings were a total surprise to everyone. The one most extensive lesion that he had was a diffuse polyposis of the intestinal tract which started one millimeter beyond the pylorus and extended to one centimeter above the internal sphincter of the rectum. In the duodenum and throughout the large bowel there was not a centimeter of gut uninvolved. In the jejunum and ileum one occasionally struck patches of mucosa a few centimeters in length without a polyp, but I think there was no segment so great as three centimeters in length that was uninvolved. All of these appeared as shaggy soft benign polyps with the exception of one which was evidently at the site of the ampulla of Vater. The common bile duct was about the size of a normal duodenum. The duodenum itself was swollen by the polyposis to at least three times the normal diameter and when we traced down the common bile duct we could find no opening into the duodenum. On cutting through the region of the ampulla we found a single small area of firm hard cancer; in other words, one of these polyps, and only one, had become malignant, completely shutting off the common bile duct. The bile ducts throughout the liver were dilated and as we got up to the smaller bile ducts they were filled with creamy yellow pus. There was a diffuse cholangitis which enlarged the liver to about 2500 grams. There were no metastases from the tumor. His various other lesions as far as we could make out were multiple exostoses, hard, ivory-like nodules usually close to the epiphyseal line, perfectly typical of benign exostoses. The skin lesions were just what he said they were, wens; he had about fifty of them.

DR. ALLEN: Was no rectal examination or proctoscopy done on this patient?

DR. SOLEY: Rectal examination was done, but no proctoscopy.

A PHYSICIAN: What was the banana-like mass across the abdomen?

DR. MALLORY: That was a distended gall bladder.

DR. MCKITTRICK: What made it go across the abdomen?

DR. MALLORY: The liver was very much distorted on account of the extreme scoliosis and was lying almost vertically instead of horizontally; the gall bladder lay at a right angle to the long axis of the liver.

We examined the spine carefully in gross—we have no sections—and we were entirely unable to find any evidence of Pott's disease. I think the scoliosis was a functional one in all probability.

DR. KRANES: I forgot to mention that he had carried large bundles of newspapers on his shoulder for some time and that perhaps had something to do with the scoliosis.

A PHYSICIAN: What were the glands in his neck?

DR. MALLORY: Those we did not examine.

DR. A. V. BOCK: How about the edema?

DR. MALLORY: The kidneys were perfectly normal. I think both the anemia and the edema were probably due to failure of absorption of substances through this extraordinarily abnormal intestinal canal; hardly a single portion was normal.

A PHYSICIAN: What about his weight?

DR. MALLORY: He had lost over thirty pounds in weight. The diarrhea was at least at one period a fairly presenting symptom, although when he came to the hospital it was much less.

DR. ARLIE V. BOCK: You are assuming that

the polyposis was of very recent origin. He was well up until five months before he came in.

DR. MALLORY: I do not believe we have any grounds on which to base a guess as to how long he had the polyposis.

DR. MILLER: I think one can have polyposis coming on fairly young in life and yet not causing any symptoms, that is, polyposis of the small intestine.

DR. BOCK: Yes; but Dr. Mallory is assuming that the edema is due to absorption and then of course he must assume that this is a recent development.

A PHYSICIAN: I think the liver might be a cause of the edema.

DR. MALLORY: His liver might have played a rôle at the end, but the edema came on several months before the jaundice. It seems to me that persistent diarrhea, low serum protein and marked anemia would be adequate cause for the edema.

CASE 21062

PRESENTATION OF CASE

A twenty-four year old Canadian-American housewife entered complaining of severe headache.

She had been comparatively well, although never very strong, until about eight months before entry. During that month she gave birth to her first baby, a normal delivery followed by an uneventful recovery. Two and a half months before admission she failed to have her regular menstrual period and after about six weeks a physician gave her some medicine which brought on rather severe uterine bleeding. She flowed for about a week and was forced to take ergot three times a day for five days. The bleeding continued and about five weeks before entry a curettage was performed, following which the bleeding stopped. She lost about ten pounds in weight during this period and felt quite tired and worn out. She lost her appetite but did not have cough, chest pain or night sweats. Three weeks before entry she developed a moderately severe headache which was relieved by empirin. One week later the headache returned with increased severity and remained located in the occipital region. It was not relieved by codein. One week before admission the headache became unbearable and she vomited. Her neck was not stiff and she had no impairment of vision. Sodium amytal helped her somewhat, but produced increased frequency and nocturia. Five days before admission she was much worse. Her eyes were heavy and her mind irrational. She recognized only her husband. She was sent to a hospital where a lumbar puncture showed slightly cloudy fluid under high pressure with very low sugar and no bacteria. Her neck was stiff at that time and the headache, located chiefly at the base, was very severe. She continued to be irrational and was transferred to this hospital.

Her father died at the age of fifty-five of a cerebral accident. Her mother was living and well. There was no history of cancer or tuberculosis.

She had been married two years. Her husband and one child were living and well. There were no miscarriages.

She had the usual childhood diseases and an appendectomy ten years before entry.

Physical examination showed a well-developed and nourished woman lying in bed moaning and complaining of headache. She was moderately dehydrated. The neck was stiff and the pupils widely dilated. The bladder was distended. Kernig's signs were positive. The reflexes were hyperactive and equal on both sides. The blood pressure was 120/80. The heart and lungs were negative. She had numerous delusions of objects and persons but was oriented in all three spheres.

The temperature was 99.5°, the pulse 75. The respirations were 28.

Examination of the urine showed a specific gravity of 1.020 with a slight trace of albumin. The sediment was negative. The blood showed a red cell count of 5,200,000, with a hemoglobin of 75 per cent. There was a white cell count of 14,350, 85 per cent polymorphonuclears. A lumbar puncture showed an initial pressure of 750 with good dynamics. Twenty-five cubic centimeters of slightly cloudy fluid was removed. The ammonium sulphate and alcohol tests were positive. The total protein was 195 milligrams per cent, sugar 27.5, and chloride 532. There were 195 white blood cells, 125 lymphocytes and 70 polymorphonuclears. A smear stained for tubercle bacilli and bacteria was negative. A Wassermann test was doubtful. The gold sol was 0111232100.

A chest plate showed slight prominence of the lung markings in both infraclavicular regions. The film, however, was of poor quality and was insufficient evidence for a positive diagnosis.

The patient continued to have active delusions and developed urinary retention. She rapidly failed, became stuporous and died on the sixth day.

DIFFERENTIAL DIAGNOSIS

DR. JAMES B. AYER: The family history and past history give nothing which appears to bear on the present illness, nor can we directly relate the menstrual abnormalities, as stated, with the subsequent history of headaches culminating in death. One is tempted to think that absence of menstrual flow perhaps signified a malnutrition and was a conservative process. No pelvic le-

sions appear to have been demonstrated in connection with this.

The present illness appears to be definitely established three weeks before entry and four weeks before death. Headache is the earliest and most significant symptom, and its severity may well be judged by the fact that it was not relieved by codein. Stiffness of the neck together with an irrational mental status, occurring two weeks after the onset of headache, are consistent with the onset of some form of increased intracranial pressure and probably meningitis. The spinal fluid examination, carried out previous to entry and repeated by us, conclusively shows meningitis of a subacute or chronic type. The fluid is not that of an acute septic process caused by pyogenic organisms, nor is it compatible with the aseptic type of meningitis, in that the low sugar and chloride findings are those seen in bacterial meningitis. By the time she reaches our hospital, therefore, the clinical picture is one of meningitis. The spinal fluid picture corroborates this and suggests that it is a subacute type of meningitis and this is also borne out by a slight rise in temperature. In spite of the fact that tubercle bacilli were not demonstrated, one can hardly hesitate in making a diagnosis of tuberculous meningitis. It is unfortunate that the x-ray film of the lungs was unsatisfactory; however, it not infrequently happens that tuberculosis is present in the lungs as a miliary tuberculosis, and is not demonstrable at the time the films are taken. It is unusual in tuberculous meningitis not to have cranial nerve palsies, particularly with reference to the oculomotors, but the absence of cranial nerve symptoms and signs should not prevent us from making this diagnosis.

Clinical Diagnosis

Tuberculous meningitis.

Dr. James B. Ayer's Diagnosis

Tuberculous meningitis. No foci of infection demonstrated.

Anatomic Diagnoses

Tuberculous meningitis.
Acute miliary tuberculosis.
Chronic tuberculous adenitis of the bronchial glands.
Tuberculous salpingitis.
Tuberculous peritonitis.

Pathologic Discussion

Dr. Tracy B. Mallory: Dr. Ayer was quite correct in his diagnosis of tuberculous meningitis. Since this can never be the primary focus of the tuberculosis in the body he naturally looked for evidence of an older lesion elsewhere, but the record was too scant to give him any significant leads. In retrospect, it is probable that the menstrual abnormalities were actually an indication of what he was looking for, but since they were not backed up with the demonstration of definite pathology by pelvic examination they were entirely inadequate for any diagnosis. She did have a bilateral tuberculous salpingitis and also a tuberculous endometritis. One often wonders how long lesions of this type may have existed before they are discovered at operation or autopsy. In this case I think we can confidently assume that they were of less than seventeen and in fact probably less than eight months' duration. This condition is so uniformly connected with sterility that its development must be subsequent to conception and probably to the pregnancy. It would be very interesting to know what the endometrium showed at the curettage done five weeks before entry to the hospital. It seems very probable that tubercles might have been found in it at that time. The salpingitis, however, was not the origin of the tuberculous process, which proved to be a very chronic tuberculous adenitis of bronchial glands. As terminal events acute miliary tuberculosis and tuberculous peritonitis accompanied the meningitis. Naturally, if the patient had reached the hospital at an earlier stage in her illness instead of in incipient coma, the chances of demonstrating these other lesions would have been very much greater.

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THE GENE

THE search for the gene, the living structural unit which governs heredity, has just been carried a long step forward by biologists working in two different American institutions. Hitherto the gene has been invisible, because of its minute size; and has resembled the atom in that it was known by its effects alone. Within the past year, however, discoveries have been made which render visible the location of the genes within the chromosomes and which promise to reveal the genes themselves for visual study. These discoveries were revealed at the recent meetings of the Genetics Society of America by Professor T. S. Painter of the University of Texas and by Dr. C. B. Bridges of the California Institute of Technology, the men responsible for this latest advance in genetics.

While the chromosomes are usually so small that it is not possible to make out any internal detail, it has been found that in the salivary glands of the fruit fly, Drosophila, the cells possess enormous chromosomes, hundreds of times bulkier than those in the other cells of the body. These chromosomes are so large that it is easily seen that they are composed of darkly staining disks, rings, or beads of various shapes and sizes alternating with colorless bands, the exact pattern being the same in corresponding chromosomes from cell to cell or fly to fly. By treatment with x-rays, it is possible to break pieces off the chromosomes and thus to produce various changes in the appearance of the flies. By breeding tests, the geneticist can tell the position of the break with reference to the genes of the affected chromosome, and the cytologist is able to study the giant chromosomes in the salivary glands and see where the break is located with regard to the visible pattern of the chromosome. In this way, Painter and Bridges and their coworkers have been able to locate various genes in or near certain chromosomal bands of characteristic appearance. The work has advanced so far that it is already possible to map the position of a considerable number of the genes most frequently used in genetic studies.

At present it is perhaps too early to evaluate the whole significance of this discovery. These giant chromosomes with their visible bands are known so far only in the flies; fortunately, however, the fruit fly is the best known genetically of all animals and so is an ideal form for the preliminary working out of this new field. Whether it will be possible to extend this method to the study of mammalian or human genetics cannot be guessed yet. It should be noted also that none of the investigators claim to have actually seen the gene, despite newspaper report to that effect. The number of genes is probably far larger than the number of visible bands, so that the larger bands must represent several or many genes. The most that can be said at present is that the dark bands apparently indicate the position of the genes on the chromosomes; whether they are actually the genes themselves can only be decided after further study. In any case this new discovery is of extreme importance to the geneticist, ranking in value with the discovery that it is possible to produce mutations by means of x-rays.

VINYL ETHER

VINYL ether, or vinyl oxide, has recently been introduced as an inhalation anesthetic and will probably be on the open market under the name "Vinethene" before long. Before this product had been made, before it even existed except in theory, prediction was made by Leake,

from its chemical structure, that it would make a good anesthetic. It has been investigated experimentally rather thoroughly, chiefly by a group at the University of Pennsylvania, and has been used clinically in several thousand cases by a number of different clinicians.

It has been found experimentally to have very little toxic action with the exception of its action on the liver. Respiration is depressed well before circulation, as with the commonly used ethyl ether, so that at cessation of respiration the heart is still beating strongly. There is ample margin between the anesthetic and the lethal concentrations, the ratio between the two being about 1 to 2½. However, its action is so rapid and powerful that the patient may be precipitated into the fourth stage of anesthesia much more readily than with ethyl ether, and if he is so precipitated too deeply, resuscitation may be difficult. It is inflammable and forms, as does the other ether, highly explosive mixtures with the correct proportions of air, nitrous oxid, or oxygen. It has not proved a very good anesthetic for dogs, as it produces some mucus and muscular excitement. Pathological examination of the various organs of the body has failed to show any tissue changes with the exception of the liver. Here there was definite necrosis in dogs when the anesthesia was maintained for over two hours. Such necrosis did not occur in monkeys. In liver function tests both on dogs and on human beings, Bourne found little evidence of damage except where anoxemia was present, and recommends its use in obstetrics. From these pieces of experimental work it appears that, while it is quite possible to produce serious liver damage (this has, in fact, occurred clinically), under conditions favorable for its development, such as prolonged and deep anesthesia, anoxemia or deficiency of glycogen, yet if these factors are avoided the effect on the liver is no more than with ethyl ether.

Clinical trial has shown considerable diversity of results. Some observers have reported very satisfactory results, while others have found a high incidence of various unpleasant phenomena, such as excessive mucus, muscular excitement, crowing respiration with obstruction, and cyanosis. This diversity of experience may be due in part to differences in administration, since most of the poor results have been reported by those having a comparatively small experience with its use. Some of the poor results in its early use may also have been due to impurities in the drug, since, unless it is properly stabilized, it decomposes rather easily with the formation of such irritating products as formaldehyde and formic acid. The manufacturers state, however, that at the present time the drug is exceptionally pure and so well stabilized that any trouble from this source has been eliminated. Induction and recovery are very rapid, the former taking one and one-half to three minutes and the latter one-half to five minutes. Abdominal relaxation is good. It may be administered in various ways like ordinary ethyl ether, by an open method on an ordinary mask, by a closed method in a gas machine, or as an adjuvant to one of the gases, but, because of its rapid evaporation, it is preferably used by some closed method.

On the whole, it appears probable that in vinyl ether we have a valuable addition to our list of anesthetic agents. While it is not a good anesthetic for deep and prolonged anesthesias (roughly an hour) it appears very suitable where an anesthesia is wanted for a rapid induction and recovery and good relaxation.

QUESTIONABLE PUBLICITY

THERE is a common belief pervading the medical profession that doctors should exercise discretion in expressing disapproval of work done by their colleagues. Boston surgeons have, on two recent occasions, been the object of rather severe criticisms. The latest occasion was the statement reported to have been made before the Johns Hopkins University Supper Club, when an eminent physician of Boston is reported to have said, "There are an enormous number of surgical operations that ought never to have been done." *Enormous* means very many when used in this way. Very properly a surgeon in this meeting contended that, since the alleged conditions were emphasized by a Boston doctor, the criticisms must apply to Boston surgeons because he was confident that such practices were not frequent everywhere.

We hope that our Boston friend was not correctly quoted; but if he was, a definite lesson should be learned; first, that general statements affecting the ethics of an honorable group of practitioners should not be made; and secondly, if the author of these remarks has evidence to substantiate his beliefs, he should make specific charges to the American College of Surgeons, the Boston Surgical Society, the New England Surgical Society, or even, in an aggravated case, to the Board of Registration in Medicine.

It would be in order, we believe, for the Massachusetts Medical Society to ask the gentleman to give the names of any of its members who may be guilty of unethical practices, if he is able to do so. If this is merely loose talk, the doctor should at least be urged to be more careful in his utterances, not so much as a courtesy to his professional brethren, but especially to avoid creating prejudice among the laity. Some nonprofessional people are already disturbed and have raised the question as to the behavior of surgeons. We expect such accusations at legislative hearings, but not at a gathering of medical men open to reporters.

THE DEATH OF DR. THOMAS J. O'BRIEN

Notice of the death of Dr. Thomas J. O'Brien, Executive Assistant to the President of the Massachusetts Medical Society, has reached this office as we go to press. An extended notice will appear in the next issue.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Munro, Donald. A.B., M.D. Harvard University Medical School 1916. F.A.C.S. Visiting Surgeon in charge of Neurosurgery, Boston City Hospital. Assistant Professor of Neurological Surgery, Harvard Medical School. Address: Boston City Hospital, Boston, Mass. Associated with him is

Hahn, Joseph. M.D. Tufts College Medical School 1931. Assistant in Neurology, Harvard University Medical School and Tufts College Medical School. Formerly Resident in Neurosurgery, Boston City Hospital. Now, Resident in Neurology, Boston City Hospital. Address: Boston City Hospital, Boston, Mass. Their subject is "Tidal Drainage of the Urinary Bladder." Page 229.

Truesdale, Philemon E. M.D. Harvard University Medical School, 1898. F.A.C.S. Surgeon, Truesdale Hospital, Fall River, Mass. His subject is "Diaphragmatic Hernia at the Esophageal Hiatus. The Short Esophagus and Thoracic Stomach." Page 240. Address: 151 Rock Street, Fall River, Mass.

Albright, Fuller. A.B., M.D. Harvard University Medical School 1924. Instructor in Medicine, Harvard Medical School. Assistant Physician, Massachusetts General Hospital. Address: Massachusetts General Hospital, Boston, Mass. Associated with him is

Halsted, James A. A.B., M.D. Harvard University Medical School 1930. Assistant in Medicine, Massachusetts General Hospital. Address: 264 Beacon Street, Boston, Mass. Their subject is "Studies on Ovarian Dysfunction." Page 250.

Wyman, Edwin T. M.D. Tufts College Medical School 1911. Visiting Physician at the Children's Hospital, Boston and the Infants' Hospital, Boston. Consulting Pediatrician Norwood Hospital, Norwood; Leominster Hospital, Leominster; Framingham Union Hospital, Framingham and Burbank Hospital, Fitchburg. Instructor in Pediatrics, Harvard University Medical School. Address: 319 Longwood Avenue, Boston, Mass. Associated with him are

Eley, R. Cannon. M.D. University of Virginia, Department of Medicine, Charlottesville, 1925. Instructor in Department of Pediatrics and Communicable Diseases, Harvard Medical School. Associate Visiting Physician at Children's Hospital, Boston. Address: 319 Longwood Avenue, Boston, Mass. And

Bunker, John W. M. A.B., A.M., Ph.D. Professor of Physiology and Biochemistry, Massachusetts Institute of Technology. Address: Massachusetts Institute of Technology, Cambridge, Mass. And

Harris, Robert S. S.B. Research Associate, Department of Biology, Massachusetts Institute of Technology. Address: Massachusetts Institute of Technology, Cambridge, Mass. Their subject is "A Comparison of 'Yeast Milk' and Irradiated Milk in the Treatment of Infantile Rickets." Page 257.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

Thomas Almy, M.D., *Chairman*, 140 Rock Street, Fall River, Mass.

C. J. Kickham, M.D., *Secretary*, 524 Commonwealth Avenue, Boston, Mass.

WISDOM OF VAGINAL VS. RECTAL EXAMINATION IN LABOR

In recent years there seems to have been a marked tendency on the part of medical men toward rectal examination of the parturient during labor. This is particularly true of men obstetrically trained, and is a definite improvement in the care of cases during labor. It was chiefly used to ascertain the advancement of the head in the pelvis and the progress of labor, but, increasing experience has proved that it has a much wider field of usefulness.

Obviously it has eliminated one very important factor in the causation of puerperal sepsis. One does not need to employ all the technique that is used in examining a patient vaginally. It is sufficient to have the gloved finger well covered with lubricant so that there will not be produced an abrasion of the rectal mucosa on inserting the finger. The index finger of either hand is best suited to this procedure.

Examination can be conducted without pain because, during pregnancy and labor, the sphincter and levator ani muscles are softer and more dilatable. The rectovaginal wall can be easily compressed and the outlines of the cervix together with its dilatation and the presenting part can be determined with comparative ease.

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

In breech and face presentation the diagnosis is not so easy. Not infrequently it is possible, when the cervix is well thinned out, to diagnose the position of the fetus by feeling the cranial sutures or other landmarks on the presenting part. The extent of engagement can be determined because the spines of the ischia are felt without difficulty. Since the examination is practically painless and with a minimum of danger of infection, it can be done many times to determine the progress of labor.

Sometimes it is not possible to feel the presenting part by rectal examination, unless a slight amount of pressure is put on the presenting part just above the symphysis, or unless a little fundal pressure is used. Rectally one can decide about the presence of a prolapsed cord, a footling, tumors, rigid perinei and the spaciousness or not of the birth canal. Sometimes, however, in placenta previa it is necessary to examine vaginally. The majority of labors at the present time are conducted with rectal and abdominal examinations only. There are but a few men who still adhere to the vaginal method and who claim very little sepsis. Since rectal examination will furnish us with sufficient information about any given labor, with rare exceptions, and since it obviates one definite potential source of infection, it is a desirable procedure in the conduct of the average labor.

MASSACHUSETTS LEGISLATIVE NOTES

SCHEDULE OF HEARINGS

Thursday, **February 7,** at 10:30 A.M., in Room 450, State House, before the Committee on Public Health.

H. 528 provides for regulation of the practice of physicians and surgeons in certain cases.

Opposed by the Committee on State and National Legislation of the Massachusetts Medical Society.

Tuesday, **February 19,** at 10:30 A.M., in Room 450, State House, before the Committee on Public Health.

H. 60 requires the vaccination of children in private schools.

Approved by the Committee.

H. 623 is a bill making vaccination voluntary.

Opposed by the Committee.

H. 755 is a bill to prevent vaccination or inoculation with impure virus or serum and without consent.

Opposed by the Committee.

H. 1059. This is designed to define the powers and duties of the Milk Control Board.

S. 229. A resolve for an investigation of the New England Milk Producers Association.

S. 213. This relates to the construction and equipment of additions to the Westfield State Sanatorium and for caring for residents of the four western counties suffering with pulmonary tuberculosis.

H. 1045 relates to expenditures for mosquito control in Cape Cod.

H. 1157 is designed to create a board of examination and registration of chiropractors.

H. 1175 is designed to abolish the Industrial Accident Board.

H. 1679 is designed to provide additions to the Middlesex County Sanatorium.

H. 1105 is an act requiring the payment of certain hospital bills as part of the settlement of claims for personal injuries or death.

H. 602 is an act preventing the discontinuance of compensation ex parte by a member of the industrial accident board except upon the report of a physician appointed by said board.

H. 601 is an act making parents of certain minors entitled to receive workmen's compensation payable on account of injuries resulting in the death of such minors.

H. 1288 is an act relative to payments under the workmen's compensation act during the period of total incapacity.

H. 454 is an act relative to the weekly payment of wages. Hearing February 7. 10:30 A.M., Room 427, State House.

H. 455 is an act limiting the hours of labor of public employees and of women and certain minors to thirty hours per week.

H. 633 is an act authorizing the town of West Springfield to appoint a town physician for certain purposes.

H. 603 is an act to prevent the discontinuance of payments under the workmen's compensation law on ex parte hearings. Hearing February 7. 10:30 A.M., Room 427, State House.

S. 124 is an act enlarging the original and concurrent jurisdiction of the Supreme Judicial and Superior Courts to include certain suits by hospitals to reach and apply certain assets.

H. 562 is an act relative to the educational qualifications of applicants for registration in Pharmacy.

S. 268. Petition of Dwight O'Hara that the Department of Labor and Industries be authorized to pay fees to physicians for certain medical reports to said department. Hearing February 7. 10:30 A.M., Room 427, State House.

H. 521. Petition of Charles F. Hurley, Chairman of the State Board of Retirement, for legislation to regulate medical fees of physicians acting for said board in disability cases.

H. 758. Petition of Curtis M. Hilliard for investigation by a special commission (Commissioners of Public Health and Mental Diseases and others) of public health laws and practices within the Commonwealth.

H. 1157. Petition of Henry J. Kennedy for establishment of a board of examination and registration to regulate the practice of chiropractic.

S. 308. Petition of Frank Hurley that provision be made for the establishment and maintenance, in the western part of the Commonwealth, of a hospital for the treatment of cancer and for the temporary relief and treatment of persons suffering from cancer.

H. 581 is an act creating a Board of Commissioners to administer a motor vehicle insurance fund for providing compensation for injuries and deaths due to accidents caused by motor vehicles. This provides among other things that actions of contract or tort for malpractice, error or mistake against physicians, surgeons, dentists, optometrists, hospitals and sanatoria shall be commenced only within two years next after the cause of action accrues, and actions for libel shall be commenced only within one year next after the cause of action accrues.

H. 1276 is an act prohibiting the handling of tort claims by doctors.

H. 715 is an act relative to death payments under the workmen's compensation law, an amendment to chapter one hundred and fifty-two of the General Laws which is as follows:

If the dependents of an injured employee who dies while receiving disability compensation hereunder file with the department within thirty days after his death a written request for the appointment of a medical arbitration committee to determine the question as to whether the death resulted from the injury for which compensation was being paid, the department shall thereupon establish such a committee which shall consist of a physician selected by the insurer, a physician selected by the dependents of the deceased employee and a third physician selected by the department whose fee and expenses shall be allowed the same as those of an impartial physician under section nine and shall be paid in the same manner. The determination by said committee of the aforesaid question shall be binding upon the department and shall be embodied in the final decision rendered by the department or any of its members.

H. 675 is an act establishing the University of the Commonwealth of Massachusetts. The object is as follows: This university of the commonwealth of Massachusetts shall include, and have full control over, the state normal schools at Barnstable, Bridgewater, Fitchburg, Framingham, Lowell, North Adams, Salem, Westfield, and Worcester; together with the Massachusetts school of art, the Massachusetts state college, the Massachusetts nautical school, the Bradford-Durfee textile school at Fall River, the Lowell Textile school, the New Bedford textile school and the division of university extension.

MISCELLANY

THE AWARD OF THE ORDER OF THE WHITE ROSE TO DR. WILLIAM P. MURPHY

Dr. William P. Murphy was awarded the Order of the White Rose by the President of Finland with a rating of Commander of the First Rank, December 1934; made a member in December 1934 of the Kaiserlich. Leopold.-Carolin. Deutsche Akademie der Naturforscher, which was established in 1652 and is probably the oldest of our scientific societies.

HEALTH OFFICERS' MONTHLY STATEMENT OF VENEREAL DISEASES REPORTED

TREASURY DEPARTMENT—PUBLIC HEALTH SERVICE

NOVEMBER, 1934

This statement is issued monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The following reports were received from State Health Officers. The figures are preliminary and subject to correction. It is hoped that this will stimulate more complete reporting of these diseases.

State	Syphilis		Gonorrhea	
	Cases Reported During Month	Monthly Case Rates per 10,000 Population	Cases Reported During Month	Monthly Case Rates per 10,000 Population
Alabama (a)	—	—	—	—
Arizona	22	.49	217	4.79
Arkansas (c)	369	1.97	231	1.23
California	1,362	2.25	1,373	2.26
Colorado (a)	—	—	—	—
Connecticut	260	1.58	172	1.04
Delaware	203	8.42	27	1.12
Dist. of Columbia	136	2.75	131	2.65
Florida	389	2.50	42	.27
Georgia	633	2.17	369	1.27
Idaho	0	0	0	0
Illinois	1,303	1.66	1,211	1.55
Indiana	234	.71	180	.55
Iowa (c)	105	.42	182	.73
Kansas	132	.69	81	.43
Kentucky	142	.54	273	1.03
Louisiana	187	.87	133	.62
Maine	43	.54	45	.56
Maryland	739	4.44	230	1.38
Massachusetts	390	.90	596	1.38
Michigan	525	1.04	489	.97
Minnesota	289	1.11	300	1.16
Mississippi (b)				
Missouri	332	.91	190	.52
Montana (c)	61	1.13	37	.69
Nebraska	52	.37	86	.62
Nevada (a)	—	—	—	—
New Hampshire	19	.41	21	.45
New Jersey	550	1.31	261	.62
New Mexico (c)	48	1.11	42	.97
New York	5,067	3.91	1,559	1.20
North Carolina	1,234	3.77	351	1.07
North Dakota	14	.20	60	.87

Ohio (c)	785	1.15	223	.33
Oklahoma (b)				
Oregon	39	.40	60	.61
Pennsylvania	313	.32	205	.21
Rhode Island	100	1.42	47	.67
South Carolina (c)	244	1.40	314	1.80
South Dakota	7	.10	30	.43
Tennessee (c)	492	1.85	278	1.04
Texas	609	1.01	152	.25
Utah (a)	—	—	—	—
Vermont	24	.66	29	.80
Virginia (c)	245	1.00	224	.92
Washington	183	1.14	213	1.33
West Virginia (b)				
Wisconsin (d)	31	.10	170	.57
Wyoming (a)	—	—	—	—
Total	17,912	1.56	10,834	0.94

(a) Not reporting.

(b) Has been reporting regularly but no report received for current month.

(c) Incomplete.

(d) Only cases of syphilis in the infectious stage are reported.

Surveys in which all medical sources have been contacted in representative communities throughout the United States have revealed that the monthly rate per 10,000 population is 6.6 for syphilis and 10.2 for gonorrhea.

CORRESPONDENCE

A LESSON FROM A COURT EXPERIENCE

January 30, 1935.

Editor, *New England Journal of Medicine*,

An experience that I have just had might be worth something to my fellow members of the Society, because it points out a danger, it has evidently established a Court precedent, and it suggests a practical method of handling cases in the future.

Putting it simply, it was a case of a very minor injury due to an automobile collision. Much to my surprise an attorney was injected into the case, and later a physician friendly to the case. A report was immediately requested of me and promptly given. Fortunately for me, I think, no side was favored. About a month later notification came to me by telephone that the case was to be tried upon a certain date, and I was requested to hold myself subject to telephone call.

That morning before Court opened I carefully reviewed the case, refreshed my memory as to the general anatomy of the back, including the muscles, ligaments, and other structures which I wished to have pictured in my mind, as well as the general surgical aspects especially as to treatment and prognosis; this of course being routine practice before giving evidence.

Nothing was heard from the case all that day, and of course the day had been spent in anticipation of a call, with its necessary adjustments. Two weeks later the case did come up, and the same preparation had to be made.

Now as I say, about a month following the accident and about three weeks after I last saw the patient perfectly free from symptoms, the new man saw the case once, doubtless in anticipation of the case coming to trial. There was no good fortune in securing a friendly agreement on the diagnosis.

The plaintiff got a verdict much greater than the merits of the case would justify, so far as I could see.

I received a check for the medical services but none for the Court proceedings. The attorney claimed that since I did not include a charge over and above the actual medical fees for treatment, that I was entitled to no more than the amount of the fees up to the time of the trial. The plaintiff refused to pay any fee for my services as a witness, either as to fact or as to expert evidence. I had never met the attorney in the case before, although I had had some experience with him on one previous case which was settled out of Court, and it took me about nine months to get the final five dollars.

When the witness fee was refused I took the matter up personally with the presiding justice, and as a result took the case into the Small Claims Court. The case was bitterly fought, the attorney apparently determined not to lose. However, I won my case. A request for a retrial was made by the attorney, and this morning I get notice that retrial is refused. That ends the chapter so to speak.

Now what I want to point out is the danger that might have been involved should I have tried to be, in any sense, impartial to either the plaintiff in the case, or to the insurer. Sticking absolutely to my guns possibly has saved me much trouble. I might have been the one on the defense.

Then again it would appear that for the first time apparently to our knowledge is it recognized, in Court, that a physician is entitled to an expert fee. The Court asked the attorney why a physician should not be paid when he would never object to paying a plumber a large fee for expert evidence. The attorney replied that he could find no evidence of a physician ever having been paid for expert evidence, and asked if the Court wanted to take upon itself the responsibility of creating a precedent. The Court evidently did wish to do so, stating that in these cases that a contract was implied, and that the physician was entitled to a fee for expert evidence.

I have pointed out a certain danger that it might be well to avoid. It is often dangerous to attempt to be the good fellow. A strict, scientific viewpoint is always the safe road to follow, as legal entanglements may become foreign entanglements under certain conditions, and one can easily become permanently enmeshed. In dealing with attorneys keep strictly to the straight and narrow, no matter who they are.

Finally I wish to make a suggestion that has come to me from my experience in this, one of my rare

cases. Avoid giving medical reports to lawyers whom you do not personally know. You are not required, by law, to give reports. It is better not to do so at all. You have, however, the right to state plainly to your patient from the very first visit, that you will refuse to give a medical report to any attorney except one whom you know personally and have absolute confidence in, as to his fair dealing with you. You have the right to refuse to give expert evidence if you choose, and it would be better, I feel, to refuse to give expert evidence in the case where attorneys unknown to you are involved. You can be summonsed into Court. That of course is true, but would it not be a foolish thing to summons an expert into Court, as a witness of fact, when he could refuse point blank to answer questions where technical or professional problems are concerned? No fully sane lawyer would attempt that more than once, I am sure.

You are entitled to a fair minimum fee as expert, and you can collect, because a contract is definitely implied. You can refuse to give medical reports. I would suggest that you do so, except to lawyers of your own choosing as to whom you will give reports, and I am sure, if you do use that veto power, that you will stand a pretty good chance of collecting on all cases whether private or ward cases, where your patient wins his suit. It is unfortunate that we know so little about these legal matters, and it is doubly unfortunate that our *Journal* aids us so little in the common practical problems of life that are really so essential. Few of us are men of independent means, most of us are struggling along and making unnecessary mistakes.

Problems such as these should be freely discussed at our District Medical Society meetings, but they seldom are. Each one of us has problems, common to all. Our district meeting would be far more interesting and profitable to us if we discussed things, in general, rather than listened to some dry address that we know very well we could much better learn and appreciate if we read it at home seated comfortably, and at ease.

It is true that the problems that I have discussed in this letter are of deep interest to every one of us in practice, doing general work or surgical work, especially. Many of the men following other specialties probably do not share the responsibilities and disappointments, in tort cases, that these two groups do, and are not so much interested. But to the one who has cared for these cases, probably for days, weeks and often months, in the wards, or privately, and finds that he is asked to accept a small reward or get nothing at all, unless you happen to be a personal friend of the lawyer, while the lawyer always gets his full share, the whole matter is disappointing.

You have the power, however, in your own hands, to refuse strange lawyers, a report. You have that right to refuse. And if you want to be paid for your work, which most of us do, then a strict use of your power will help you very materially.

I think we have been "goops" long enough, and the sooner we, in the districts, find that we need better service in working out our financial problems, the sooner proper machinery will be provided.

Sincerely yours,

W. A. HUTTON, M.D.

525 Lynn Fells Parkway, Melrose, Mass.

ABLATION OF THE THYROID GLAND

January 29, 1935.

Editor, *New England Journal of Medicine*,

Recently, there has been much ado in the literature relative to "Total Ablation of the Thyroid" in certain forms of cardiac disease. This procedure and its elicited results are sufficiently important to merit serious attention. To those who have read only one side of the topic, O. R. Lourie's article (*Canadian M. A. J.*, Nov., 1934), describing the original work of Kocher in 1883 on "Ablation of the Thyroid with Its Disastrous After Effects," is recommended. This should be supplemented by a careful reading of Dr. Henry Christian's letter on "Total Ablation of the Thyroid" which appeared in the *Journal of the American Medical Association* dated January 5, 1935 (p. 64).

Very truly yours,

J. L. GRUND, M.D.

483 Beacon Street,
Boston, Mass.

RECENT DEATHS

A DELAYED DEATH NOTICE

GILBERT — JOHN GILBERT, M.D., of 254 Locust Street, Fall River, died in that city, November 3, 1934. Dr. Gilbert was born in England in 1847, and graduated from the University of Vermont College of Medicine in 1886.

He was, for many years, surgeon-general of the Department of Massachusetts, G. A. R., and city physician. He enlisted in the army during the Civil War, and was commander of Richard Borden post, G. A. R., of Fall River, at the time of his death.

He is survived by his widow, Mrs. Elizabeth Gilbert.

DURANT — CHARLES EDWIN DURANT, M.D., of 105 Emerson Street, Haverhill, Massachusetts, died in that city, January 19, 1935, after several months of ill health.

He was born in Epping, New Hampshire, September 3, 1862, the son of the late Charles Edwin Durant and Harriet Shepard Durant. His early education was acquired in the Epping and Haverhill public schools. He graduated from Phillips-Andover Academy in 1881 and from the Harvard Medical School in 1885. He was an interne in the Boston City Hospital. He began practice in Haverhill in 1886. For

many years he served on the staff of the Hale Hospital and the Gale Hospital.

Dr. Durant joined the Massachusetts Medical Society in 1885, was also a Fellow of the American Medical Association, and was one of the group of 1000 surgeons at the inauguration of the American College of Surgeons. He was a member of the board of appeals during the World War and after the Massachusetts State Guard was organized, he was commissioned as captain, and later promoted to rank of major with assignment to the 16th Regiment, Infantry, as chief medical officer. After the 16th Regiment was disbanded, Dr. Durant was transferred to the 11th Regiment with headquarters in Boston.

His civic interests were memberships on the school board and water board, having served as chairman for four years. Dr. Durant was a Mason.

He is survived by his widow, Mrs. Marion F. (Foster) Durant; two daughters, Mrs. A. A. C. Malcolm and Mrs. Mark Packard, Jr.; and a grandson, all of Haverhill.

FULLER — Daniel Hunt Fuller, M.D., a former member of the Massachusetts Medical Society, died in Philadelphia, February 1, 1935. He was assistant to the Executive Officer of the Massachusetts Department of Mental Diseases before moving to Philadelphia in 1914. He was recognized as a psychiatrist of standing and for the past ten years was Chairman of the American Psychiatric Society's Committee on Nursing.

Dr. Fuller was chief of the clinic of mental and nervous diseases of the Pennsylvania Hospital.

KNOWLES — Charles Augustus Knowles, M.D., of 695 Adams Street, Dorchester, with an office at 520 Commonwealth Avenue, died at his home, January 31, 1935. He was a member of the visiting staffs of the Boston City, the Cambridge, and St. Elizabeth's Hospitals.

Dr. Knowles was born in Boston in 1904, the son of the late William J. and Catherine C. Knowles. He was educated in the public schools and Tufts College and graduated from Tufts College Medical School in 1927. He served as house officer at the Boston City Hospital and joined the Massachusetts Medical Society in 1930.

He is survived by his widow, Mrs. Agnes Fitzgerald Knowles; a daughter, Clare; and four brothers, William Knowles and Albert Knowles, both of Boston, Joseph Knowles of Stoughton and Edward Knowles of Fitchburg.

DOBSON — Clarence Henry Dobson, M.D., of 29 Williston Road, Brookline, Mass., died at his home, February 1, 1935, after a brief illness. He was born in Lynn in 1869, the son of John Martin and Joanna Short Dobson. After graduating from the Lynn public schools, he matriculated at the Hahnemann Medical College and Hospital of Philadelphia, graduating therefrom in 1897. He began practice in Ardmore, Pa., and later moved to Conway, Mass., and from there enlisted in the army in August, 1917. He served with Base Hospital No. 60 at Bazoilles, France. Returning to this country after the war, he settled in Brookline.

He joined the Massachusetts Medical Society in 1921, and was also a Fellow of the American Medical Association.

NOTICES

THE AMERICAN NEISSERIAN MEDICAL SOCIETY

January 30, 1935.

The American Neisserian Medical Society was founded on June 12, 1934. It is dedicated to the promotion of knowledge in all that relates to the gonococcus and gonococcal infections, that there may be attained improvement in the management of gonorrhea and a reduction in its prevalence. There are 115 charter members and the officers are as follows:

Dr. Edward L. Keyes, New York, Honorary President.

Executive Committee:
Dr. J. Dellinger Barney, Boston, President,
Dr. P. S. Pelouze, Philadelphia, Vice-President,
Dr. A. L. Clark, Oklahoma City,
Dr. Walter Clarke, New York,
Dr. R. D. Herrold, Chicago,
Dr. N. A. Nelson, Boston,
Dr. Oscar F. Cox, Jr., Boston, Secretary-Treasurer.

The society plans to carry out the following program:

A. The scrutiny of the management of gonorrhea in both male and female.
B. Clinical and laboratory research in the diagnosis, medical and social pathology, and the treatment of gonorrhea.
C. Dissemination among the medical profession and the public of authoritative information concerning gonorrhea.

Membership is limited to:

A. Residents of the United States or its territories, Canada or Mexico.
B. Graduates of a medical school recognized by the American Medical Association.
C. Those who are engaged in some phase of the management of gonorrhea.

Invitation to membership is extended to all qualified physicians who desire to work for improvement in the management of gonorrhea. Application blanks can be obtained from the undersigned.

Oscar F. Cox, Jr., M.D., *Secretary.*
475 Commonwealth Avenue,
Boston, Mass.

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, February 15, 12-1, Cheever amphitheatre. Dr. William R. Morrison, Associate Professor of Surgery, will present:

1. A case of obstructing duodenal ulcer.
2. A case of acute pancreatitis associated with gall stones.
3. In addition, certain surgical cases will be presented for discussion.

Physicians and medical students are invited.

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, February 14, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

REMOVAL

WALTER O. BLANCHARD, M.D., announces the removal of his office to 465 Centre Street, Newton, Massachusetts.

REPORT AND NOTICES OF MEETINGS

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The New England Ophthalmological Society met Tuesday evening, January 15, at the Massachusetts Eye and Ear Infirmary. The meeting was called to order by Dr. Hugo Riemer.

The first business was the election of officers which resulted as follows: President, Dr. James Regan; Vice-President, Dr. Edwin A. Goodall; Secretary-Treasurer, Dr. Benjamin Sachs; Recording Secretary, Dr. Trygve Gundersen. Dr. Regan then took charge of the meeting.

Dr. Rowland presented a twelve year old boy who apparently had been unable to rotate the eyes laterally since birth. The boy was undersized and underdeveloped for his age, due probably to underactivity of the anterior pituitary. The case was discussed by the guest of the evening, Professor Bielschowsky, who demonstrated partial paralysis of the external recti, which were very weak. In his opinion this was a case of congenital defects of the external recti, probably with inelastic fibrous bands. Here there was no pathological convergence as would be expected in acquired bilateral sixth nerve paralysis.

Dr. Casten presented a twenty-eight year old woman who six months ago had a sudden onset of diplopia within 24 hours following childbirth. The diplopia was homonomous for distance and crossed for near. Discussion was concerning the differential diagnosis of convergent spasm and divergent paralysis, with opinion favoring the former.

Dr. Alexander Marble discussed the studies of blood fat in two cases of Lipemia Retinalis. Both cases were in young boys entering the hospital in diabetic coma, markedly acidotic. The blood fat values were markedly elevated, seven and one-half per cent in one case and fourteen per cent in the other, the normal value being seven-tenths per cent. The blood cholesterol was also elevated in each case. Under active treatment the blood fats returned to normal; about five per cent disappeared in the first twelve hours, the fat presumably being stored in the body. Lipemia Retinalis is a relatively uncommon condition occurring usually in young males in acidosis from uncontrolled diabetes where the blood fats exceed three per cent.

Professor Bielschowsky presented a paper, "Congenital and Acquired Anomalies in Fusion." Factors in deficiency of fusion may be disturbances in either the sensory or motor apparatus. There is a small group of patients with diplopia due to a slight squint who have a condition known as "horror fusion". In this condition the correcting prism does not give single vision, but merely transfers the diplopia from crossed to uncrossed or vice versa.

Professor Bielschowsky presented at some length a patient who apparently had this condition. This was a twenty-five year old man who developed a convergent strabismus following a blow on the head when a child. At the age of fourteen he was operated upon to correct the squint and was given glasses. One year ago the patient suddenly developed diplopia with headache and loss of weight. Several operations on the extra-ocular muscles were performed without relief, and all attempts to correct the diplopia by refraction and prisms were ineffective. It was then discovered that the patient had anisicoria. Fusion could only be maintained for short periods of time and with the head held in a very abnormal position. By using many consecutive anisicoric corrections, the head was gradually brought down to the primary position while still retaining fusion. With the use of anisicoric lenses the patient now has fusion and depth perception.

ROBERT BRECK BRIGHAM HOSPITAL

CLINICAL MEETING

There will be a clinical meeting on "Chronic Arthritis" at the Robert Breck Brigham Hospital, 125 Parker Hill Avenue, on Wednesday evening, February 20, at 8 o'clock. There will be case presentations and discussions by the Staff. Physicians and medical students are cordially invited.

JOHN G. KUHNS, M.D.,
Secretary to Staff.

MASSACHUSETTS MEMORIAL HOSPITALS

There will be a meeting of the Surgical Section in the Ladies' Aid Room (former nurses' dining room), Talbot Memorial, 82 East Concord Street, on Friday, February 8, 1935, at 12 noon.

A résumé of his service will be given by Dr. Harry J. Lee.

MILO C. GREEN, M.D., *Secretary.*

SPRINGFIELD ACADEMY OF MEDICINE

On Tuesday evening, February 12, at the Springfield Academy of Medicine, Dr. R. G. Leland, Director, Bureau of Medical Economics, American Medical Association, will speak on "Current Medical Problems," and Dr. Nathan B. Van Etten, one of the signers of the minority report of the "Committee on the Costs of Medical Care," will speak on "An Economic Program for 1935."

JAMES A. SEAMAN, M.D., *Secretary.*

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The February meeting will be held at the Boston Medical Library, Friday night, February 15, 1935, at 8:15 P.M.

SCIENTIFIC SESSION

"Tumors of the Kidney." Dr. E. Ross Mintz, Urological Service of the Massachusetts General Hospital.

RICHARD DRESSER, M.D., *Secretary.*

THE TRUDEAU SOCIETY

A meeting of the Trudeau Society will be held at the Prendergast Preventorium, Mattapan, Massachusetts, on February 12, 1935, at 4 P.M.

Dr. Alton S. Pope will read a paper on the "Epidemiology of Tuberculosis." Dr. John B. Hawes, 2nd, will discuss "The Scope, Function, and Results of Preventorium Care."

Dr. Henry D. Chadwick, State Health Commissioner, will open the discussion.

MOSES J. STONE, M.D., *Secretary.*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 19, 1935, 12 noon. The speaker will be Thomas J. Richards, M.D., Assistant Visiting Surgeon, Boston City Hospital. His subject will be "Athletic Injuries." All physicians are cordially invited to attend both lecture and luncheon.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance) Tuesday evening, February 12, at 8:15 P.M.

PROGRAM

Presentation of Cases.

The Significance of Changes in Plasma Volume as Determined by the Dye Method, by Dr. Magnus I. Gregersen.

The Toxicity of Two Dyes Used in Plasma Volume Determinations, by Dr. John G. Gibson, II.

MARSHALL N. FULTON, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, FEBRUARY 11, 1935

Tuesday, February 12—

1:30 P.M. Radio Program. WEEI. "The Trends in Sewage Disposal." (Continued.)

†2:30-4 P.M. Ward visit, Massachusetts Eye and Ear Infirmary.

4 P.M. The Trudeau Society. Prendergast Preventorium, Mattapan.

†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.

4:30 P.M. Radio Program. WBZ. "Ear, Nose and Throat Diseases."

8:15 P.M. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance).

Wednesday, February 13—

3 P.M. New England Dermatological Society. Massachusetts General Hospital.

Thursday, February 14—

*8:30 A.M. Lecture and Clinic on Heart Disease by Dr. Christian. Peter Bent Brigham Hospital.

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference. Children's Hospital.

*3:30 P.M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital.

†4:30 P.M. Surgical Clinic. Children's Hospital Amphitheatre.

Friday, February 15—

†12 M. Clinical meeting of Children's Medical Staff, Massachusetts General Hospital. Ether Dome.

12-1 P.M. Boston University School of Medicine Surgical Clinic at the Boston City Hospital, Cheever Amphitheatre.

5 P.M. Radio Program. WEEI. "Safe Water Supplies." "Nuisances."

8:15 P.M. New England Roentgen Ray Society. Boston Medical Library, 8 Fenway.

Saturday, February 16—

*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

Sunday, February 17—

4 P.M. Harvard University. (Medical School, Building D, Longwood Avenue, Boston.) Free lecture. "Diabetes." Dr. H. F. Root.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

February 7—Faulkner Hospital Clinical Meeting will be held at 5 P.M.

February 8—William Harvey Society will meet in the Auditorium of the Beth Israel Hospital, Boston, at 8 P.M.

February 8—Massachusetts Memorial Hospitals. See notice elsewhere on this page.

February 12—Harvard Medical Society. See notice elsewhere on this page.

February 12—The Trudeau Society. See notice elsewhere on this page.

February 12—Springfield Academy of Medicine. See notice elsewhere on this page.

MASSACHUSETTS DIETETIC ASSOCIATION

February 12—Tuesday, 8 P.M. "Diabetic Children," Dr. Priscilla White, Joslin Diabetic Unit.

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

February 13—New England Dermatological Society will meet at the Massachusetts General Hospital at 3 P.M.

February 14—Clinic at the Peter Bent Brigham Hospital. See page 277.

February 15—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 277.

February 15—New England Roentgen Ray Society. See page 278.

February 19—South End Medical Club. See page 278.

February 20—Robert Breck Brigham Hospital, Clinical Meeting. See page 277.

February 20—Brookfield Medical Club will meet at the Hampshire House, Ware, Mass.

March-May—International Medical Postgraduate Courses in Berlin. Programs and further particulars are obtainable from the Berlin Academy for Medical Postgraduate Training, Berlin NW7, Robert Koch-Platz 7 (Kaiserin Friedrich-Haus). German as well as foreign doctors may attend the courses.

March 11, 12, 13—Surgeons to meet in Jacksonville, Florida (Southeastern Surgical Congress). See page 83, issue of January 10.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 27-29 inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. The American delegation to the Congress will sail from New York on August 8 and visit London, Amsterdam, The Hague and Paris and, optionally, Budapest. Physicians interested in the Congress or in the medical tour in conjunction with it may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Annual Meeting will be held in May. Time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of March and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 13, 1935—Wakefield.

May 8, 1935—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

February 26, 1935—Hotel Kenmore, 8 P.M. The Use of Amniotic Fluid in Abdominal Surgery. Dr. Herbert L. Johnson.

March 26, 1935—Fernald School for Feeble-Minded, Waverley. Details to be announced.

May, 1935—Annual Meeting. Date, time and place to be announced.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March—Plymouth County Hospital.

April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 27, 1935—Clinical Meeting at the Boston Lying-In Hospital.

April 24, 1935—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

March 13, 1935—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10, 1935—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8, 1935—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOKS RECEIVED FOR REVIEW

Transactions of the American Association of Genito-Urinary Surgeons. Forty-sixth Annual Meeting held at Hot Springs, Va., May 14, 15, and 16, 1934. Volume XXVII. 438 pp. Saint Paul and Minneapolis: The Bruce Publishing Company.

Standard Classified Nomenclature of Disease. Compiled by The National Conference on Nomenclature of Disease. Edited by H. B. Logie. 870 pp. New York: The Commonwealth Fund. $3.50.

Medical Tactics and Logistics. Colonel Gustavus M. Blech and Colonel Charles Lynch. 205 pp. Springfield and Baltimore: Charles C Thomas. $4.00.

How to Practice Medicine. Henry W. Kemp. 156 pp. New York: Paul B. Hoeber, Inc. $2.50.

The Clinical Aspects of Visceral Neurology. With special reference to the surgery of the sympathetic nervous system. W. K. Livingston. 254 pp. Springfield and Baltimore: Charles C Thomas. $5.00.

Clio Medica. Edited by E. B. Krumbhaar. XV. French Medicine. M. Laignel-Lavastine, and M. Raymond Molinery. 187 pp. New York: Paul B. Hoeber, Inc. $2.50.

La Renaissance de la Médecine Humorale. Auguste Lumière. 204 pp. Lyon: Imprimerie Léon Sézanne.

Annual Report of the Surgeon General of the Public Health Service of the United States. For the fiscal year 1934. 143 pp. Washington: United States Government Printing Office. $.75.

Poliomyelitis. A handbook for physicians and medical students. Based on a study of the 1931 epidemic in New York City. John F. Landon and Lawrence W. Smith. With a section on the orthopedic after care of the disease by Garry DeN. Hough, Jr. 275 pp. New York: The Macmillan Company. $3.00.

Aids to Psychiatry. W. S. Dawson. Third Edition. 318 pp. Baltimore: William Wood & Company. $1.50.

Aids to Embryology. Richard H. Hunter. Second Edition. 172 pp. Baltimore: William Wood & Company. $1.25.

BOOK REVIEWS

Surgical Applied Anatomy. Sir Frederick Treves, Bart. Philadelphia: Lea & Febiger. 1934.

It is always a great satisfaction to welcome a new edition of Treves's Applied Anatomy, that marvel among textbooks which for fifty years has maintained unabated the prestige which it established immediately upon its first publication. It was in 1883 that this book first made its appearance. Subsequent editions by the author, and later by Sir Arthur Keith and Mr. Colin Mackenzie, have maintained the standard of the original with gradual modification to suit the progress of the times but without essential alteration in the spirit and purpose of the work. This ninth edition, edited by Mr. C. C. Choyce, M.D. (Edin.), F.R.C.S. (Eng.), aims likewise "to preserve the relationship between the anecdotal surgical anatomy of Treves's time and that of to-day." Though a good deal of the original text has disappeared in the interests of anatomical and surgical detail, enough has been retained to preserve the historical interest and essential temper of the original. The total number of pages has been increased by only eighteen over the seventh edition of 1917. The total number of illustrations has been increased by twenty-one; but of these only sixty-six are in color, as against seventy-four in 1917. To students and teachers alike, this book bids fair to continue for years a cherished and esteemed *vade mecum*, which may be said of it in the true sense that it is a compendium which can go everywhere in the possessor's pocket. Like the schoolmaster in the *Deserted Village*, the marvel still grows — that it can carry in such relatively small compass all the valuable material which it contains.

Prolapsus du Rectum. Carrasco. 196 pp. Paris: Masson et Cie. 35 fr.

A paper-covered edition of one hundred and ninety-six pages with forty-one illustrations, the book is an extensive review of the literature on this subject since the complete work of Lenormant in 1903, together with numerous case reports. Carrasco derives his own material from the clinic of Henri Hartmann in Paris, and Hartmann has prefaced the book. The author discusses, in the twenty-eight pages of Part I, the etiology, pathology, and symptomatology of rectal prolapse. Part II, which comprises the bulk of the book, deals entirely with the numerous and varied methods of treatment; non-surgical, semi-surgical, and surgical, many of which are familiar to us. He reviews the numerous sclerosing injection solutions which have been used, and likewise the use of the cautery to produce inflammatory adhesions of the rectum to its surrounding tissues. The procedure of Thiersch, in which a wire, silk thread, or elastic ligature is inserted with a curved needle around the anal ring subcutaneously and left in place for three to twelve months, is reviewed, and case records are given in connection with most of the procedures. Eleven different abdominal suspension operations are considered in detail, and in the following chapter are outlined nine perineal procedures, among which that of Cuneo and Seneque is described and illustrated in detail.

Carrasco has also taken up special procedures to be employed in cases of prolapse of the new anus after resection of the rectum for cancer and, finally, he has discussed the association of rectal with genital prolapse, and the surgical treatment of this complication.

An extensive bibliography of seventeen pages is appended. The book represents a great deal of work on the part of the author, and work well done. It is complete, easily readable, and a real contribution to the subject.

Surgical Clinics of North America. October, 1934. Volume 14, Number 5. Lahey Clinic Number. 260 pp. Philadelphia: W. B. Saunders Company. $12.00 per year.

This issue opens with a group of interesting clinics on gastroenterological topics such as cancer of the stomach, total gastrectomy, hemorrhage in peptic ulcer, selection of operation and integration of medical and surgical therapy in peptic ulcer. Cattell describes a simple method of closing small intestine fistulae by placing in the lumen of the intestine a small button which is tied to a similar button on the surface of the abdomen.

Lahey discusses the problem of painless jaundice. If the gall bladder is to be used for an anastomosis, he much prefers to make the connection to the jejunum rather than to either the stomach or duodenum. Clute and Swinton now believe that it is impossible to predict which cases of obstructive jaundice will be subject to postoperative hemorrhage, although bleeding is more frequent where the sedimentation rate is rapid.

The technic of intratracheal anesthesia is completely and clearly described by Sise. Photographs are generously used, especially to illustrate the use of the direct laryngoscope in passing the tracheal catheter.

Overholt demonstrates a very simple, effective method of securing tidal irrigation in the closed drainage treatment of empyema.

No collection of papers from the Lahey Clinic would be complete without some new light on thyroid problems. The aspects here considered are diagnosis of hyperthyroidism, operations in the presence of cardiac disease, goiter in patients over sixty, and myasthenia gravis and goiter.

Cattell discusses kraurosis vulvae for which he advises excision of the involved area. He also reports a modification of their technic for plastic closure following excision of a pilonidal sinus.

These with several other interesting clinics make this number, one of genuine value for any surgeon.

Review of Dr. F. J. Cotton's Chapter on Fractures in Lewis' Surgery. Second Edition.

An endeavor to make a scientific monograph presentable as a literary production has long been something to be desired in our textbook literature. After all there would appear to be nothing incompatible in this, for there is no reason why a doctor should not say what he has to say in a succinct and graphic style—if he can. A fillet of sole may come off the grill perfectly cooked and yet the tartar sauce that is served with it takes away none of whatever nutritive value it possesses, and at the same time it makes it more palatable to many tastes. It is, therefore, refreshing to read the section on fractures in Dr. Dean Lewis' second edition, written by Dr. Frederic J. Cotton, than whom there is no one in the country better qualified to write authoritatively, and certainly no one has ever done so in a more acceptable manner than in the article under consideration.

Possessed of an incisive and picturesque method of presentation in verbal communications he has not departed from it in this revision of his original text. This has resulted in a clearance of unnecessary verbiage and a departure from a more academic style, so generally affected when one formally sets out to commit his thoughts to paper. The values allotted to different features, whether of treatment or diagnosis, and very obviously based upon a not inconsiderable experience emphasize the second impression, which is perhaps enhanced by the above-mentioned style of presentation. The effect of such statements is invariably to help one to concentrate on the essentials of the situation and not be too fussy about refinements of technique where they are not the essentials. Emphasis is laid upon the fact that the goal to be reached is primarily a functional restitution, by the most direct and safest methods that experience has taught us to employ. If this can be accomplished by a "restitutio ad integrum," so much the better but if not, and the first object is achieved, there can no blame attach.

What the reviewer would again emphasize, and commend, is the production of a textbook article which any doctor may read and at the end may very likely exclaim, "It was a pleasure to have read this." At the same time that the style attracts there is no feeling that there is any lack of "up to the minute," authoritative statement. One gets the impression that conservatism is the guiding principle and yet due latitude is allowed to radicalism under the proper circumstances, which circumstances are clearly defined.

Periodic Fertility and Sterility in Woman. A natural method of birth control. Professor Hermann Knaus. 162 pp. Vienna: Wilhelm Maudrich. $6.00.

Previous to the publication of Dr. Knaus's book on "Periodic Fertility and Sterility in Woman" there have been two small books in English on this same subject. The first, "The Rhythm" by Leo J. Latz, M.D., and the second, "Conception Period of Women" by Kyusaku Ogino, M.D. The first of these was for popular use only. The second combined a résumé of the literature on this subject and particularly the scientific work of Dr. Ogino with directions for the use of the method by patients.

Dr. Knaus's book is published entirely for the medical profession. While it is somewhat polemic in tone, it does give a most satisfactory and complete review of all the experimental and truly scientific work on the physiology of reproduction which has led to the promulgation of the new point of view of the periodic variability of woman's fertility. The idea which has been tenaciously held for many years that woman can conceive at any time in her menstrual cycle finds no corroboration whatever in the mass of data which have been accumulated within recent years on mammalian reproduction including that of the primates. Knaus's argument seems incontrovertible that reproduction in man follows similar fixed rules. The only doubt is as to the invariability of these rules. After reading Dr. Knaus's work no one can doubt that ovulation occurs only once in the month and that fertilization, even allowing for the life of the spermatozoa in the woman's secretions, and for the slight doubt as to how long the egg remains capable of being fertilized, can only occur over a period of two or three days at the most. Many, many instances are quoted by Dr. Knaus of women who have used this knowledge as a successful means of birth control. What we lack is a controlled group of some five hundred couples who have used this method of avoiding the fertile time in the month to control conception, or of utilizing it when they have wanted children. A study of this sort should cover two or three years and should report with the greatest detail any apparent failures of the method. This knowledge is needed before we can recommend the use of this method to the individual patient whose health makes it essential that pregnancy be avoided. But in spite of this lack of human statistics we can thoroughly recommend the book and we believe that time will demonstrate the correctness of the general thesis that there is a natural and harmless method of regulating reproduction.

The Principles of Therapeutics. The Abraham Flexner Lectures. Series Number Three. Francis Richard Fraser. 135 pp. Baltimore: The Williams & Wilkins Company.

This volume constitutes series number three of the Abraham Flexner Lectures at Vanderbilt University. Previous to the writing of the work the author had been associated with the compilation of the new British Pharmacopoeia in which an attempt was made to test the therapeutic value of all substances listed.

Aside from the first chapter on the historical development of therapeusis there is little that cannot be found in a good textbook of medicine.

That Heart of Yours. By S. Calvin Smith. 212 pp. Philadelphia and London: J. B. Lippincott and Company. $2.00.

In the foreword of this book the author makes it clear that its purpose is "to supplement and amplify the information which physicians give to a heart patient". It is directed at the patient and is meant for his perusal rather than for the physician, although many of the latter may also derive profit from its reading. It is written in a very light vein and can be read very rapidly. Throughout its chapters one is impressed by a most optimistic point of view. This is particularly adapted to produce a proper psychological effect upon one afflicted with heart disease, even if the cold facts sometimes tell a different story. The value of this volume to the patient need not be affected even if some statements like the following are at least open to question; "but rheumatic fever can be anticipated and its course and after effects either modified or averted by early recognition of its advent" and "that probably sixty-five per cent of the attacks of angina pectoris that appear in men who are in their forties result from sexual overindulgence". There also seems to be an undue emphasis on the importance of infected teeth as a cause of heart disease.

If one believes that popularizing medical information to the lay public is worth while, this volume can serve at least as a buffer to the ever-increasing dread of heart disease that has stricken our country.

L'Eczéma du Nourrisson. Par les docteurs M. Péhu et R. Aulagnier. 174 pp. Paris: Gauthier-Villars, Éditeur, 1934. 25 fr.

In this little book the authors consider eczema as a distinct entity to be treated not only from the local point of view but as originating on the background of hereditary allergy. The symptoms are discussed in great detail and the various types are delimited so sharply as to be almost artificial. The admission that the various types may occur together tends to obscure this sharp definition into types. There is much emphasis on the allergic factors and relationships. Stress is laid on laboratory tests, the variations in the serum protein of the blood, eosinophilia, the various types of skin tests by intradermal or patch tests, or by passive transfer. The intradermal tests, especially by an enterococcus, are especially considered. Hypotheses on the nature of infantile eczema are discussed, and the hereditary allergic nature is again brought out. Attention is called to the sudden unexplainable deaths occurring in a small proportion of cases of infantile eczema. The authors state that general treatment ought to be given first place, and that bacterial vaccines ought to be systematically employed in all cases of eczema as the method of choice to alter the allergic state. Numerous formulae for external applications are given, but care should be taken that these are not "aggressive or brutal". A bibliography of references, mostly in the French literature, is appended.

Annals of the Pickett-Thomson Research Laboratory. Monograph XVI, Part II. Influenza. By David Thomson and Robert Thomson. 1557 pp. London: Baillière, Tindall and Cox. $17.50.

Part I of this monograph, which was reviewed in the July 12 issue of the *Journal*, now appears to have been, in the words of Capt. Henry, "only the beginning, folks, only the beginning"; for here are some 400,000 additional words on the subject, including a bibliography of over 4500 references. The authors may well congratulate themselves for their "health has been maintained in spite of the stupendous effort".

The present volume is devoted to the complications and sequelae, their bacteriology, the pathology, epidemiological data, prevention and treatment of influenza. In turning over this great dust-pile many interesting but useless articles have been uncovered. The authors' own too infrequent views, appraisals and summaries are the most instructive sections of the work. They tell us again that the outstanding relationship between influenza and encephalitis is their mutual association with filterable viri; that Pfeiffer's bacilli, the strepto- and pneumococci are variable, secondary or complicating organisms; that vaccines, if they are of any prophylactic value, are protective against the pulmonary complications; that views regarding the effect of influenza on the course of pulmonary tuberculosis are discordant; thus, and much more.

The essential weakness of the material rests in the fact that it so often and necessarily harks back to the data of 1918. We knew as little about influenza then as we did about pernicious anemia. Perhaps some day our present knowledge of influenza will take on the quaint chaotic look of the anemia literature of that decade. That day will dawn when and if we learn to understand and deal with the filterable viri. Then this pile will become history, and good history, too.

Murrell's What to Do In Cases of Poisoning. By P. Hamill. Beginning the Fourteenth Edition of "What to Do in Cases of Poisoning" by the late Dr. W. Murrell. 208 pp. New York: Paul B. Hoeber, Inc. $1.50.

It is probably true that no man will read a very large book on poisons unless he is particularly interested in toxicology, but this little work is well worth the hour required to go through it. The fact that it has gone through thirteen previous editions emphasizes its utility. The section on criminal toxicology deserves more than a hasty glance. It contains all the essential facts, arranged in easy reference form. Many of the clinical conditions are placed in outline form with the suggestion of the type of substance most likely to cause a particular group of signs and symptoms. It should be in the emergency room of every hospital.